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Search Results - Record(s) 1 through 4 of 4 returned.☐ 1. Document ID: US 20020132225 A1

L1: Entry 1 of 4

File: PGPB

Sep 19, 2002

PGPUB-DOCUMENT-NUMBER: 20020132225

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020132225 A1

TITLE: Compositions and methods for prolonging survival of chilled platelets

PUBLICATION-DATE: September 19, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Stossel, Thomas P.	Belmont	MA	US	
Hartwig, John H.	Jamaica Plain	MA	US	
Wagner, Denisa D.	Wellesley	MA	US	

US-CL-CURRENT: 435/4; 435/7.2

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KIMC	Draw Desc	Image
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☐ 2. Document ID: US 20020040008 A1

L1: Entry 2 of 4

File: PGPB

Apr 4, 2002

PGPUB-DOCUMENT-NUMBER: 20020040008

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020040008 A1

TITLE: Method for treating and preventing atherosclerosis

PUBLICATION-DATE: April 4, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Wagner, Denisa D.	Wellesley	MA	US	
Johnson, Robert C.	Sparta	NJ	US	

US-CL-CURRENT: 514/41; 424/130.1, 514/54, 514/8

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KIMC	Draw Desc	Image
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☐ 3. Document ID: US 20020031508 A1

L1: Entry 3 of 4

File: PGPB

Mar 14, 2002

PGPUB-DOCUMENT-NUMBER: 20020031508

PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020031508 A1

TITLE: Methods for diagnosing and treating hemostatic disorders by modulating
P-selectin activity

PUBLICATION-DATE: March 14, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
<u>Wagner, Denisa D.</u>	Wellesley	MA	US	
Andre, Patrick	Jamaica Plain	MA	US	
Hartwell, Daqing W.	Brookline	MA	US	
Hrachovinova, Ingrid	Jamaica Plain	MA	US	

US-CL-CURRENT: 424/94.63; 424/145.1, 514/12

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	NAME	Draw Desc	Image
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☐ 4. Document ID: US 5807745 A

L1: Entry 4 of 4

File: USPT

Sep 15, 1998

US-PAT-NO: 5807745
DOCUMENT-IDENTIFIER: US 5807745 A

TITLE: Method of inhibiting PADGEM-mediated or ELAM-1-mediated leukocyte adhesion
using an inhibitor comprising a Le.sup.x core component

DATE-ISSUED: September 15, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Furie; Bruce	Wellesley	MA		
Furie; Barbara C.	Wellesley	MA		
Larsen; Eric	Lebanon	NH		
Palabrica; Theresa	Quincy	MA		
Sajer; Susan A.	Brookline	MA		
<u>Wagner; Denisa D.</u>	Wellesley	MA		

US-CL-CURRENT: 435/375; 436/501, 436/63

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	NAME	Draw Desc	Image
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Term	Documents
WAGNER-DENISA\$	0
WAGNER-DENISA-D.USPT,PGPB.	4
WAGNER-DENISA\$.USPT,PGPB.	4
(WAGNER-DENISA\$).USPT,PGPB.	4

Display Format:

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-
- ☐ 1. [20020098563](#). 02 Mar 01. 25 Jul 02. Novel core 2 b ta-1,6-N-acetylglycosaminyltransferase gene. Korczak, Bozena, et al. 435/193; 435/320.1 435/325 435/69.1 536/23.2 C12N009/10 C07H021/04 C12N005/06.
-
- ☐ 2. [20020076833](#). 01 Aug 01. 20 Jun 02. Analysis of biological samples utilizing a coated solid phase. Henry, Michael R., et al. 436/518; G01N033/543.
-
- ☐ 3. [20020061863](#). 02 Nov 01. 23 May 02. Novel, specific inhibitors of acute and chronic inflammation. Uppugunduri, Srinivas. 514/49; 514/269 514/738 A61K031/7115 A61K031/513 A61K031/047.
-
- ☐ 4. [20020058034](#). 12 Jul 01. 16 May 02. Inhibition of differentiation of cytotoxic T-cells by P-selectin ligand (PSGL) antagonists. Manjunath, Narasimhaswamy, et al. 424/144.1; 514/12 514/54 A61K039/395 A61K038/17 A61K031/726.
-
- ☐ 5. [20020045202](#). 28 Feb 01. 18 Apr 02. Novel core 2 beta-1,6-N-acetylglycosaminyltransferase gene. Korczak, Bozena, et al. 435/15; 435/193 435/252.3 435/320.1 435/325 536/23.2 C12N009/10.
-
- ☐ 6. [20020040008](#). 18 Jun 01. 04 Apr 02. Method for treating and preventing atherosclerosis. Wagner, Denisa D., et al. 514/41; 424/130.1 514/54 514/8 A61K039/395 A61K031/715 A61K038/16.
-
- ☐ 7. [20020037840](#). 23 Mar 01. 28 Mar 02. Novel P-selectin glycoprotein ligand (PSGL-1) binding protein and uses therefor. Lorenz, Meike, et al. 514/8; 435/325 435/6 435/69.1 530/395 536/23.5 C12Q001/68 A61K038/16 C07H021/04 C12P021/02 C12N005/06.
-
- ☐ 8. [20020025923](#). 18 May 01. 28 Feb 02. Novel selection ligands. Rosen, Steven D., et al. 514/8; 530/395 A61K038/17 C07K014/435.
-
- ☐ 9. [20010046970](#). 22 Jun 01. 29 Nov 01. Inhibition of selectin binding. Nagy, Jon O., et al. 514/53; 514/54 A61K031/726 A61K031/715.
-
- ☐ 10. [20010036931](#). 27 Apr 01. 01 Nov 01. Inhibition of cell-cell binding by lipid assemblies. Nagy, Jon O., et al. 514/53; A61K031/7016.
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Term	Documents
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
ARTERIOSCLERORI	0
PSGL\$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
((PSGL\$) AND (ATHEROSCLEROSIS OR ARTERIOSCLERORIS)).USPT,PGPB.	24

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-
- ☐ 11. [6492332](#). 11 Sep 00; 10 Dec 02. Irrigation solution and methods for inhibition of tumor cell adhesion, pain and inflammation. Demopoulos; Gregory A., et al. 514/12; 514/217 514/226.2 514/25 514/254.06 514/259.1 514/263.1 514/266.1 514/280 514/288 514/317 514/327 514/353 514/356 514/397 514/413 514/415 514/509 514/619 514/654 514/680. A61K038/00 A61K031/70 A61K031/55 A61K031/54 A61K031/495 A61K031/505.
-
- ☐ 12. [6395882](#). 03 Feb 99; 28 May 02. Selectin ligands. Rosen; Steven D., et al. 530/395; 530/350. C07K014/705.
-
- ☐ 13. [6380371](#). 13 Sep 99; 30 Apr 02. Endoglycan: a novel protein having selectin ligand and chemokine presentation activity. Sasseti; Christopher M., et al. 536/23.1; 530/350 530/380 536/23.5. C07H021/04 C07H001/00.
-
- ☐ 14. [6365715](#). 01 Nov 99; 02 Apr 02. Human cardiac/brain tollid-like protein. Arleth; Anthony J, et al. 530/350; 530/399. C07K014/435 C07K014/475.
-
- ☐ 15. [6299897](#). 15 Nov 99; 09 Oct 01. Inhibition of selectin binding. Nagy; Jon O., et al. 424/443; 424/450 514/23 514/25 514/53 514/54 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K009/70 A61K031/715.
-
- ☐ 16. [6254852](#). 16 Jul 99; 03 Jul 01. Porous inorganic targeted ultrasound contrast agents. Glajch; Joseph L, et al. 424/9.52; A61B008/00.
-
- ☐ 17. [6235309](#). 27 Feb 98; 22 May 01. Inhibition of cell-cell binding by lipid assemblies. Nagy; Jon O., et al. 424/450; 514/25 514/42 514/53 514/54 514/61. A61K009/127.
-
- ☐ 18. [6124267](#). 20 Apr 98; 26 Sep 00. O-glycan inhibitors of selectin mediated inflammation derived from [PSGL-1](#). McEver; Rodger P., et al. 514/25; 514/54 514/62 536/17.2 536/18.7. A61K031/70.
-
- ☐ 19. [6123923](#). 18 Dec 97; 26 Sep 00. Optoacoustic contrast agents and methods for their use. Unger; Evan C., et al. 424/9.52; 424/450 424/9.1 424/9.2 424/9.3 424/9.6 514/410. A61K049/00 A61K049/22.
-
- ☐ 20. [6008017](#). 16 Dec 97; 28 Dec 99. Human cardiac/brain tollid-like protein. Arleth; Anthony J, et al. 435/69.1; 435/320.1 435/325 435/6 536/23.5. C12N015/12.
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Term	Documents
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
ARTERIOSCLERORI	0
PSGL\$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
((PSGL\$) AND (ATHEROSCLEROSIS OR ARTERIOSCLERORIS)).USPT,PGPB.	24

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Search Results - Record(s) 21 through 24 of 24 returned.

☐ 21. 5985852. 16 Feb 99; 16 Nov 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/54; 424/450 514/23 514/25 514/53 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K031/715 A61K009/127 C07H001/00.

☐ 22. 5977080. 08 Jan 98; 02 Nov 99. Sulfated disaccharide inhibitors of selectins, methods for synthesis and therapeutic use. Rosen; Steven D., et al. 514/25; 514/53 514/61 536/123.13 536/124 536/18.5 536/4.1. A61K031/70 C07H015/00.

☐ 23. 5962422. 28 Feb 97; 05 Oct 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/25; 435/7.1 435/7.2 514/42 514/53 514/54 514/61. A61K031/70 G01N033/53.

☐ 24. 5783693. 23 Aug 95; 21 Jul 98. Methods for synthesizing sulfated disaccharide inhibitors of selectins. Bertozzi; Carolyn, et al. 536/124; 536/123.13 536/18.5 536/4.1. C07H001/00 C07H015/00.

Term	Documents
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
ARTERIOSCLERORI	0
PSGL\$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
((PSGL\$) AND (ATHEROSCLEROSIS OR ARTERIOSCLERORIS)).USPT,PGPB.	24

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-
- ☐ 1. [20020127691](#). 27 Nov 01. 12 Sep 02. Highly purified mocoarhagin, a cobra venom protease, polynucleotides encoding same and related proteases, and therapeutic uses thereof. Boodhoo, Amechand, et al. 435/226; 435/320.1 435/325 435/69.1 536/23.2 C12N009/64 C07H021/04 C12P021/02 C12N005/06.
-
- ☐ 2. [20020061863](#). 02 Nov 01. 23 May 02. Novel, specific inhibitors of acute and chronic inflammation. Uppugunduri, Srinivas. 514/49; 514/269 514/738 A61K031/7115 A61K031/513 A61K031/047.
-
- ☐ 3. [20020040008](#). 18 Jun 01. 04 Apr 02. Method for treating and preventing atherosclerosis. Wagner, Denisa D., et al. 514/41; 424/130.1 514/54 514/8 A61K039/395 A61K031/715 A61K038/16.
-
- ☐ 4. [20020037840](#). 23 Mar 01. 28 Mar 02. Novel P-selectin glycoprotein ligand (PSGL-1) binding protein and uses therefor. Lorenz, Meike, et al. 514/8; 435/325 435/6 435/69.1 530/395 536/23.5 C12Q001/68 A61K038/16 C07H021/04 C12P021/02 C12N005/06.
-
- ☐ 5. [20020031508](#). 17 May 01. 14 Mar 02. Methods for diagnosing and treating hemostatic disorders by modulating P-selectin activity. Wagner, Denisa D., et al. 424/94.63; 424/145.1 514/12 A61K038/48 A61K039/395 A61K038/17.
-
- ☐ 6. [20020025923](#). 18 May 01. 28 Feb 02. Novel selection ligands. Rosen, Steven D., et al. 514/8; 530/395 A61K038/17 C07K014/435.
-
- ☐ 7. [20010046970](#). 22 Jun 01. 29 Nov 01. Inhibition of selectin binding. Nagy, Jon O., et al. 514/53; 514/54 A61K031/726 A61K031/715.
-
- ☐ 8. [20010036931](#). 27 Apr 01. 01 Nov 01. Inhibition of cell-cell binding by lipid assemblies. Nagy, Jon O., et al. 514/53; A61K031/7016.
-
- ☐ 9. [6492332](#). 11 Sep 00; 10 Dec 02. Irrigation solution and methods for inhibition of tumor cell adhesion, pain and inflammation. Demopulos; Gregory A., et al. 514/12; 514/217 514/226.2 514/25 514/254.06 514/259.1 514/263.1 514/266.1 514/280 514/288 514/317 514/327 514/353 514/356 514/397 514/413 514/415 514/509 514/619 514/654 514/680. A61K038/00 A61K031/70 A61K031/55 A61K031/54 A61K031/495 A61K031/505.
-
- ☐ 10. [6413936](#). 30 Oct 96; 02 Jul 02. Glycomimetics as selectin antagonists and pharmaceuticals having antiinflammatory activity. Schmidt; Wolfgang, et al. 514/23; 514/8 514/9 536/1.11 549/200 549/356 562/459 585/275. A61K031/70.
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Term	Documents
RESTENOSIS.USPT,PGPB.	8198
RESTENOSES.USPT,PGPB.	113
PSGL\$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
((PSGL\$) AND (RESTENOSIS)).USPT,PGPB.	21

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-
- ☐ 11. [6413760](#). 18 Feb 98; 02 Jul 02. Highly purified mocoarhagin cobra venom protease polynucleotides encoding same and related proteases and therapeutic uses thereof. Boodhoo; Amechand, et al. 435/226; 435/252.3 435/320.1 435/325 536/23.1 536/23.2. C12N009/64 C12N015/57.
-
- ☐ 12. [6395882](#). 03 Feb 99; 28 May 02. Selectin ligands. Rosen; Steven D., et al. 530/395; 530/350. C07K014/705.
-
- ☐ 13. [6380371](#). 13 Sep 99; 30 Apr 02. Endoglycan: a novel protein having selectin ligand and chemokine presentation activity. Sassetti; Christopher M., et al. 536/23.1; 530/350 530/380 536/23.5. C07H021/04 C07H001/00.
-
- ☐ 14. [6365715](#). 01 Nov 99; 02 Apr 02. Human cardiac/brain tolloid-like protein. Arleth; Anthony J, et al. 530/350; 530/399. C07K014/435 C07K014/475.
-
- ☐ 15. [6299897](#). 15 Nov 99; 09 Oct 01. Inhibition of selectin binding. Nagy; Jon O., et al. 424/443; 424/450 514/23 514/25 514/53 514/54 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K009/70 A61K031/715.
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- ☐ 16. [6254852](#). 16 Jul 99; 03 Jul 01. Porous inorganic targeted ultrasound contrast agents. Glajch; Joseph L, et al. 424/9.52; A61B008/00.
-
- ☐ 17. [6235309](#). 27 Feb 98; 22 May 01. Inhibition of cell-cell binding by lipid assemblies. Nagy; Jon O., et al. 424/450; 514/25 514/42 514/53 514/54 514/61. A61K009/127.
-
- ☐ 18. [6197752](#). 05 Sep 96; 06 Mar 01. Glycomimetics as selectin antagonists and pharmaceuticals having antiinflammatory activity prepared therefrom. Schmidt; Wolfgang, et al. 514/23; 536/1.11 536/124 536/18.7. A61K031/70 C07H001/00.
-
- ☐ 19. [6008017](#). 16 Dec 97; 28 Dec 99. Human cardiac/brain tolloid-like protein. Arleth; Anthony J, et al. 435/69.1; 435/320.1 435/325 435/6 536/23.5. C12N015/12.
-
- ☐ 20. [5985852](#). 16 Feb 99; 16 Nov 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/54; 424/450 514/23 514/25 514/53 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K031/715 A61K009/127 C07H001/00.
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Term	Documents
RESTENOSIS.USPT,PGPB.	8198
RESTENOSES.USPT,PGPB.	113
PSGL\$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
((PSGL\$) AND (RESTENOSIS)).USPT,PGPB.	21

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☐ 21. 5962422. 28 Feb 97; 05 Oct 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/25; 435/7.1 435/7.2 514/42 514/53 514/54 514/61. A61K031/70 G01N033/53.

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Term	Documents
RESTENOSIS.USPT,PGPB.	8198
RESTENOSES.USPT,PGPB.	113
PSGL\$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
((PSGL\$) AND (RESTENOSIS)).USPT,PGPB.	21

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Term	Documents
P-SELECTIN.USPT,PGPB.	792
P-SELECTINS.USPT,PGPB.	74
PADGEM.USPT,PGPB.	257
PADGEMS	0
GMP-140.USPT,PGPB.	425
GMP-140S	0
GMP140.USPT,PGPB.	119
GMP140S	0
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
((P-SELECTIN' OR PADGEM OR 'GMP-140' OR 'GMP140') SAME (ATHEROSCLEROSIS OR ARTERIOSCLERORIS OR RESTENOSIS)).USPT,PGPB.	70

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Search:

L6

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DATE: Friday, May 23, 2003 [Printable Copy](#) [Create Case](#)

Set Name Query

side by side

Hit Count Set Name

result set

DB=USPT,PGPB; PLUR=YES; OP=ADJ

<u>L6</u>	('p-selectin' or padgem or 'gmp-140' or 'gmp140') same (atherosclerosis or arterioscleroris or restenosis)	70	<u>L6</u>
<u>L5</u>	('p-selectin' or padgem or 'gmp-140' or 'gmp140') and (atherosclerosis or arterioscleroris or restenosis)	390	<u>L5</u>
<u>L4</u>	(psgl\$) and (atherosclerosis or arterioscleroris)	24	<u>L4</u>
<u>L3</u>	(psgl\$) and (restenosis)	21	<u>L3</u>
<u>L2</u>	(psgl\$) same (restenosis)	0	<u>L2</u>
<u>L1</u>	wagner-denisa\$	4	<u>L1</u>

END OF SEARCH HISTORY

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Term	Documents
"P-SELECTIN GLYCOPROTEIN LIGANDS".USPT,PGPB.	0
PSGL\$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
"PSGL1/MIGG.SUB.2B".USPT,PGPB.	2
(L6 AND (PSGL\$ OR 'P-SELECTIN GLYCOPROTEIN LIGANDS')).USPT,PGPB.	15

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US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
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Search:

L7

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DATE: Friday, May 23, 2003 [Printable Copy](#) [Create Case](#)

Set Name Query

side by side

Hit Count Set Name

result set

DB=USPT,PGPB; PLUR=YES; OP=ADJ

<u>L7</u>	L6 and (psgl\$ or 'p-selectin glycoprotein ligand\$')	15	<u>L7</u>
<u>L6</u>	('p-selectin' or padgem or 'gmp-140' or 'gmp140') same (atherosclerosis or arterioscleroris or restenosis)	70	<u>L6</u>
<u>L5</u>	('p-selectin' or padgem or 'gmp-140' or 'gmp140') and (atherosclerosis or arterioscleroris or restenosis)	390	<u>L5</u>
<u>L4</u>	(psgl\$) and (atherosclerosis or arterioscleroris)	24	<u>L4</u>
<u>L3</u>	(psgl\$) and (restenosis)	21	<u>L3</u>
<u>L2</u>	(psgl\$) same (restenosis)	0	<u>L2</u>
<u>L1</u>	wagner-denisa\$	4	<u>L1</u>

END OF SEARCH HISTORY

[Generate Collection](#)[Print](#)**Search Results - Record(s) 1 through 10 of 15 returned.**

-
- ☐ 1. [20030082143](#). 12 Jun 02. 01 May 03. Receptor-mediated gene delivery using bacteriophage vectors. Larocca, David, et al. 424/93.2; 435/456 514/44 A61K048/00 C12N015/86.
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Term	Documents
"P-SELECTIN GLYCOPROTEIN LIGAND\$".USPT,PGPB.	0
PSGL\$	0
PSGL.USPT,PGPB.	84
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PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
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PSGL1.USPT,PGPB.	5
"PSGL1/MIGG.SUB.2B".USPT,PGPB.	2
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- ☐ 11. [6448083](#). 26 Feb 99; 10 Sep 02. Receptor-mediated gene delivery using bacteriophage vectors. Larocca; David, et al. 435/456; 435/320.1. C12N015/64 C12N015/63.
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Term	Documents
"P-SELECTIN GLYCOPROTEIN LIGANDS".USPT,PGPB.	0
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PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
"PSGL1/MIGG.SUB.2B".USPT,PGPB.	2
(L6 AND (PSGL\$ OR 'P-SELECTIN GLYCOPROTEIN LIGAND\$')).USPT,PGPB.	15

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Term	Documents
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PSGL\$	0
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PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
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PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
"PSGL1/MIGG.SUB.2B".USPT,PGPB.	2
(L6 AND (PSGL\$ OR 'P-SELECTIN GLYCOPROTEIN LIGANDS')).USPT,PGPB.	15

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result set

DB=USPT,PGPB; PLUR=YES; OP=ADJ

<u>L7</u>	L6 and (psgl\$ or 'p-selectin glycoprotein ligand\$')	15	<u>L7</u>
<u>L6</u>	('p-selectin' or padgem or 'gmp-140' or 'gmp140') same (atherosclerosis or arterioscleroris or restenosis)	70	<u>L6</u>
<u>L5</u>	('p-selectin' or padgem or 'gmp-140' or 'gmp140') and (atherosclerosis or arterioscleroris or restenosis)	390	<u>L5</u>
<u>L4</u>	(psgl\$) and (atherosclerosis or arterioscleroris)	24	<u>L4</u>
<u>L3</u>	(psgl\$) and (restenosis)	21	<u>L3</u>
<u>L2</u>	(psgl\$) same (restenosis)	0	<u>L2</u>
<u>L1</u>	wagner-denisa\$	4	<u>L1</u>

END OF SEARCH HISTORY

14 S2
9 S4
S5 2 S2 AND S4
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S6 2 RD S5 (unique items)
? t s6/3/all

6/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13115159 BIOSIS NO.: 200100322308
Alterations in platelet thrombus formation, leukocyte recruitment, and
intimal hyperplasia in P-selectin-deficient mice after transluminal
femoral artery injury.
AUTHOR: Smyth Susan S(a); Reis Ernane D; Fallon John T; Gordon Ron; Collier
Barry S(a)
AUTHOR ADDRESS: (a)Medicine, Mount Sinai School of Medicine, New York, NY**
USA
JOURNAL: Blood 96 (11 Part 1):p813a November 16, 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of
Hematology San Francisco, California, USA December 01-05, 2000
SPONSOR: American Society of Hematology
ISSN: 0006-4971
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

6/3/2 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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124135703 CA: 124(11)135703f PATENT
Method using agents inhibiting interaction between P-selectin??? and a
P-selectin ligand for treating and preventing atherosclerosis
INVENTOR(AUTHOR): Wagner, Denisa D.; Johnson, Robert C.
LOCATION: USA
ASSIGNEE: Center for Blood Research, Inc.
PATENT: PCT International ; WO 9533484 A1 DATE: 951214
APPLICATION: WO 95US6940 (950601) *US 253663 (940603) *US 377798 (950124)
PAGES: 35 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A;
A61K-038/02B; A61K-038/16B; A61K-031/70B DESIGNATED COUNTRIES: CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC;
NL; PT; SE
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24623 RESTENOSIS
136871 ATHEROSCLEROSIS
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24623 RESTENOSIS
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restenosis and atherosclerosis

24623 RESTENOSIS
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Processing

303 S9
3756683 P
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84 P(W)SELECTION?
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? t s11/3/all

11/3/1 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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10863398 EMBASE No: 2000345938
Roles of **P-selectin** in inflammation, neointimal formation,
and vascular remodeling in balloon-injured rat carotid arteries
Hayashi S.-I.; Watanabe N.; Nakazawa K.; Suzuki J.; Tsushima K.; Tamatani
T.; Sakamoto S.; Isobe M.
Dr. M. Isobe, Dept. of Cardiovascular Medicine, Tokyo Medical and Dental
University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519 Japan
AUTHOR EMAIL: isobemi.med3@med.tmd.ac.jp
Circulation (CIRCULATION) (United States) 03 OCT 2000, 102/14
(1710-1717)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24
? t s11/7/all

11/7/1 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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10863398 EMBASE No: 2000345938

Roles of **P-selectin** in inflammation, neointimal formation, and vascular remodeling in balloon-injured rat carotid arteries
Hayashi S.-I.; Watanabe N.; Nakazawa K.; Suzuki J.; Tsushima K.; Tamatani T.; Sakamoto S.; Isobe M.

Dr. M. Isobe, Dept. of Cardiovascular Medicine, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519 Japan

AUTHOR EMAIL: isobemi.med3@med.tmd.ac.jp

Circulation (CIRCULATION) (United States) 03 OCT 2000, 102/14 (1710-1717)

CODEN: CIRCA ISSN: 0009-7322

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 24

Background - **P-selectin** plays key roles in mediating inflammation through promoting adherence of leukocytes to activated platelets and endothelium. This process is one of the initial events in **atherosclerosis** and **restenosis** after coronary angioplasty.

Methods and Results - Using a rat balloon-injury model, we examined the role of **P-selectin** in vascular inflammatory processes. In the acute phase, immunohistochemistry revealed that **P-selectin** was intensely expressed on both activated platelets covering the denuded segment and endothelial cells of the inflamed adventitial small vessels. Treatment with an anti-**P-selectin** monoclonal antibody (MAb) for 8 consecutive days significantly inhibited neointimal formation at day 14 (42% inhibition; $P < 0.05$), and this effect persisted at day 56 (40% inhibition; $P < 0.01$) compared with the control group. Vascular shrinking accompanying adventitial fibrosis was also attenuated at day 56. Inhibition of both neointimal formation and vascular shrinking resulted in the lumen area of the anti-**P-selectin** treatment group being approx. eq.3 times larger at day 56 than that of the control group. Accumulation of CD45-positive leukocytes in the developing neointima, media, and adventitia at day 8 was significantly inhibited by treatment with the anti-**P-selectin** MAb. Scanning electron microscopy demonstrated that anti-**P-selectin** treatment resulted in a less thrombogenic surface of the arterial intima, which featured a pseudoendothelial appearance at day 14 after injury. Conclusions - These results suggest that inhibition of **P-selectin**-mediated leukocyte recruitment prevents the development of neointimal formation, adventitial inflammation, and vascular shrinking and promotes pseudoendothelialization by luminal smooth muscle cells. This treatment thus beneficially affects vascular remodeling after balloon injury in rats.

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S1	25	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHER-OSCLER?)
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S4	9	RD S3 (unique items)
S5	2	S2 AND S4
S6	2	RD S5 (unique items)
S7	3360	RESTENOSIS AND ATHEROSCLEROSIS
S8	495	S7 AND REVIEW?
S9	303	RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10	0	S9 AND P(W)SELECTION?
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Processing

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250085 GLYCOPROTEIN
293792 LIGAND
739 P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND
162724 ATHEROSCLER?
S1 25 (PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND
(ATHEROSCLER?)

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S2 14 RD S1 (unique items)
? t s2/7/all

2/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13115159 BIOSIS NO.: 200100322308

Alterations in platelet thrombus formation, leukocyte recruitment, and
intimal hyperplasia in P-selectin-deficient mice after transluminal
femoral artery injury.

AUTHOR: Smyth Susan S(a); Reis Ernane D; Fallon John T; Gordon Ron; Collier
Barry S(a)

AUTHOR ADDRESS: (a)Medicine, Mount Sinai School of Medicine, New York, NY**
USA

JOURNAL: Blood 96 (11 Part 1):p813a November 16, 2000

MEDIUM: print

CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of
Hematology San Francisco, California, USA December 01-05, 2000

SPONSOR: American Society of Hematology

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: Platelet activation at the site of vascular injury, such as
occurs after **atherosclerotic** plaque rupture or following
percutaneous intervention, results in platelet-neutrophil interactions,
which contribute to local thrombosis, downstream microcirculatory events,
and systemic inflammation. The initial interaction of activated platelets
with neutrophils is mediated by platelet P-selectin binding to neutrophil
PSGL-1. To investigate the role of platelet-neutrophil interactions
in response to arterial injury, we performed transluminal, wire-induced
injury to the femoral artery of wild-type C57B1/6 mice (n=26) and
P-selectin -/- mice (n=26). The femoral arteries of anesthetized male
mice aged 8-10 weeks were injured by passing a 0.25 mm wire in the lumen
of the femoral artery three times, and the mice were euthanized by
perfusion fixation 1 h or 4 weeks after injury. 1 h after injury in
wild-type mice, platelets were found adherent to the blood vessel wall
and neutrophils were attached to the platelets. As viewed by TEM and SEM,
the platelet layer varied between 1 and approx 3 platelets thick, and
many of the platelets in contact with the wall were spread and at least
partially degranulated. 1 h after injury of P-selectin -/- mice, the
platelet layer appeared less compact and appeared to extend further into
the lumen; moreover, more of the platelets appeared to retain their
granules. There was a striking decrease in leukocyte attachment to the
platelets. Four weeks after injury, the neointimal area in P-selectin -/-
mice (2,100 +/- 900 μm^2) was significantly smaller than in the wild-type
mice (10,200 +/- 2,100 μm^2) (p=0.004). These results indicate that
P-selectin is required for neutrophil recruitment to platelets lining the

vessel wall 1 h after injury and P-selectin deficiency protects mice from developing intimal hyperplasia 4 weeks after injury. Moreover, in the P-selectin -/- mice, the platelets depositing on the damaged vessel appeared to be less activated and to extend further into the lumen, suggesting that P-selectin may play a role in platelet activation and platelet thrombus formation. Our data support the possibility that antagonists to P-selectin may decrease intimal hyperplasia and clinical restenosis after percutaneous vascular interventions in humans.

2/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13092476 BIOSIS NO.: 200100299625
Chemokines immobilized on early **atherosclerotic** endothelium mediate monocyte arrest via VLA-4.
AUTHOR: Huo Yuqing(a); Weber Christian; Ley Klaus(a)
AUTHOR ADDRESS: (a)University of Virginia, Health Science Center, Charlottesville, VA, 22908**USA
JOURNAL: FASEB Journal 15 (4):pA584 March 7, 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: In reconstituted models using cultured endothelial cells or plate surfaces coated with chemokines and adhesion molecules, leukocyte arrest can be triggered by chemokines such as IL-8, MIP-2, GRO-alpha, RANTES and SDF-1alpha. Here, we investigate which chemokines immobilized on the spontaneous early **atherosclerotic** lesions can contribute to the arrest of rolling monocytes in **atherosclerosis**. This study was performed on a novel ex vivo model, in which monocyte roll and arrest on lesion-prone sites in carotid arteries from apoE-/-, but not control mice mainly through P-selectin/PSGL-1 and VLA-4/VCAM-1 (Huo et al., Circ. Res. 87: 146-152, 2000). Monocyte arrest in this model is blocked by 55% by pertussis toxin, by 37% with the CCR-1, 3 and 5 inhibitor, metRANTES, and by 42% with the CXCR-2 inhibitor, 8-73 GRO-alpha, but not by the CCR-2 inhibitor, 9-79 MCP-1. RANTES, KC (mouse GRO-alpha) and MCP-1 are expressed on the endothelium of arteries from apoE-/-, but not wild-type mice, and antibody to KC block arrest by 42%. Perfusing KC or RANTES through carotid arteries with early **atherosclerotic** lesions can further increased the number of arresting monocyte, while perfusing MCP-1 had no effect. Blockade of VLA-4/VCAM-1 but not CD18/ICAM-1 dramatically inhibited the arrest of monocyte both under physiological and KC or RANTES perfused conditions. These results suggest that KC and RANTES immobilized on early **atherosclerotic** lesions activate VLA-4 on monocytes to mediate efficient monocyte arrest on the luminal surface of lesion prone arteries of apoE-/- mice. Surprisingly, MCP-1 does not function as an arrest chemokines under physiologic conditions. Our findings clarify the molecular mechanism involved in monocyte recruitment to **atherosclerotic** lesions and suggest potential new approaches to curbing the development of **atherosclerosis** lesions.

2/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12864535 BIOSIS NO.: 200100071684
Complex roles of P-selectin and von Willebrand factor in inflammation and hemostasis.

AUTHOR: Wagner D D(a); Andre P(a); Denis C V(a); Hartwell D W(a);
Hrachovinova I(a); Methia N(a)
AUTHOR ADDRESS: (a)The Center for Blood Research, Department of Pathology,
Harvard Medical School, Boston, MA**USA
JOURNAL: Journal of Submicroscopic Cytology and Pathology 32 (3):p333
July, 2000
MEDIUM: print
CONFERENCE/MEETING: XIth International Vascular Biology Meeting Geneva,
Switzerland September 05-09, 2000
ISSN: 1122-9497
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

2/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12058582 BIOSIS NO.: 199900339101
Direct demonstration of P-selectin- and VCAM-1-dependent mononuclear cell
rolling in early **atherosclerotic** lesions of apolipoprotein
E-deficient mice.
AUTHOR: Ramos Carroll L; Huo Yuqing; Jung Unsu; Ghosh Shukti; Manka David R
; Sarembock Ian J; Ley Klaus(a)
AUTHOR ADDRESS: (a)Department of Biomedical Engineering, Health Sciences
Center, University of Virginia, Charlottes**USA
JOURNAL: Circulation Research 84 (11):p1237-1244 June 11, 1999
ISSN: 0009-7330
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Apolipoprotein E-deficient (ApoE^{-/-}) mice develop
atherosclerotic lesions throughout the arterial tree, including the
carotid bifurcation. Although the expression of adhesion molecules such
as ICAM-1, vascular cell adhesion molecule-1 (VCAM-1), and P-selectin on
endothelium that overlie **atherosclerotic** plaques has been
implicated in monocyte recruitment to developing lesions, monocyte
adhesion in **atherosclerotic** vessels has not been observed directly.
To investigate which adhesion molecules may be important in monocyte
adhesion to **atherosclerotic** lesions, an isolated mouse carotid
artery preparation was developed and perfused with mononuclear cells. We
show rolling and attachment of the human monocytic cell line U937 and the
mouse monocyte-macrophage cell line P388D1 in carotid arteries from 10-
to 12-week-old ApoE^{-/-} and C57BL/6 wild-type mice fed a Western-type diet
(21% fat wt/wt) for 4 to 5 weeks. No rolling was observed in carotid
arteries from C57BL/6 or BALB/c wild-type mice fed a chow diet and little
was observed in BALB/c mice fed a Western-type diet. This model
represents early lesion development as shown by minimal macrophage
infiltration in the intima of carotid arteries from ApoE^{-/-} mice fed a
Western-type diet. Rolling was observed at shear stresses that were
characteristic of the low-shear recirculation zone near the carotid
bifurcation. Mononuclear cell attachment and rolling were significantly
inhibited by monoclonal antibody blockade of P-selectin or its leukocyte
ligand **P-selectin glycoprotein ligand-1**. Rolling
velocities increased after monoclonal antibody blockade of mononuclear
cell alpha4-integrin or VCAM-1, which indicates that alpha4-integrin
interacting with VCAM-1 stabilizes rolling interactions and prolongs
monocyte transit times.

2/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

11715101 BIOSIS NO.: 199800496832
P-selectin and MAC-1 mediate monocyte rolling and adhesion to ECM-bound platelets under flow conditions.
AUTHOR: Kuijper P H M(a); Tores H I Gallardo; Houben L A M J; Lammers J-W J ; Zwaginga J J; Koenderman L
AUTHOR ADDRESS: (a)Dep. Pulmonary Dis., Room G.03.550, Univ. Hosp. Utrecht, Heidelberglaan 100, 3584 CZ, Utrecht**Netherlands
JOURNAL: Journal of Leukocyte Biology 64 (4):p467-473 Oct., 1998
ISSN: 0741-5400
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Accumulation of monocyte-derived foam cells in focal areas of the **atherosclerotic** (A.S.-) lesion is one of the key events in early atherogenesis. Using a flow model for the damaged vessel wall, we examined the ability of ECM-bound platelets to induce monocyte tethering and adhesion. Whereas, ECM-proteins alone induced monocyte adhesion only at low shear stresses (< 100 mPa), ECM-bound platelets induced monocyte rolling and adhesion at shear stresses up to 240 mPa. Studies with specific antibodies showed that monocyte adhesion to platelets was mainly mediated by P-selectin and monocyte **PSGL-1** (maximum inhibition 90%). beta2-Integrin blocking CD18 and CD11b antibodies partly inhibited the arrest of rolling cells. Antibodies against other adhesion molecules such as LFA-1, PECAM-1, and beta1-integrins had no effect. Even sparsely adhered platelets (apprx 10% coverage of the surface) already strongly supported monocyte tethering. In conclusion, activated platelets present on ECM are a powerful adhesive substrate for monocyte recruitment under flow conditions.

2/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

11486174 BIOSIS NO.: 199800267506
Adhesion of monocytes to vascular cell adhesion molecule-1-transduced human endothelial cells. Implications for atherogenesis.
AUTHOR: Gerszten Robert E; Lim Yaw-Chyn; Ding Han T; Snapp Karen; Kansas Goeffrey; Dichek David A; Cabanas Carlos; Sanchez-Madrid Francisco; Gimbrone Michael A Jr; Rosenzweig Anthony; Luscinskas Francis W(a)
AUTHOR ADDRESS: (a)Vascular Res. Div., Brigham and Women's Hosp., 221 Longwood Avenue, Boston, MA 02115**USA
JOURNAL: Circulation Research 82 (8):p871-878 May 4, 1998
ISSN: 0009-7330
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To study the role of vascular cell adhesion molecule-1 (VCAM-1) in monocyte recruitment and atherogenesis, we constructed a recombinant adenovirus, AdRSVrVCAM-1, carrying the rabbit VCAM-1 cDNA. We have previously shown that AdRSVrVCAM-1-transduced human umbilical vein endothelial cells (HUVECs) support the adhesion of CD4+ CD45RO+ memory T lymphocytes under laminar flow conditions. We now demonstrate that AdRSVrVCAM1-transduced HUVECs support the adhesion of peripheral blood monocytes at a shear stress of 1.5 dyne/cm^2 . Although VCAM-1 supported only firm adhesion of lymphocytes, it was able to mediate monocyte rolling, firm adhesion, and transmigration when expressed in the context of otherwise unactivated vascular endothelium. VCAM1-transduced HUVECs supported the adhesion of as many as 4-fold more monocytes than T cells under laminar flow. The greater monocyte adhesion was explained at least in part by leukocyte-leukocyte interactions (secondary adhesions), which were not seen with T cells. These secondary monocyte interactions were specifically blocked by monoclonal antibodies to L-selectin and

P-selectin glycoprotein ligand-1. These data demonstrate that VCAM-1 expressed in the context of unactivated vascular endothelium supports the adhesion of the leukocyte populations present in **atherosclerotic** plaque and may contribute to the predominance of monocytes over lymphocytes.

2/7/7 (Item 7 from file: 5)
DIALOG(R)File 5: Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09156353 BIOSIS NO.: 199497164723
Structure/function studies of **P-selectin glycoprotein ligand**.
AUTHOR: Barone Karen M; Pittman Deborah; Shaw Gray
AUTHOR ADDRESS: Genetics Inst. Inc., 87 Cambridge Park Drive, Cambridge, MA 02140**USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p290 1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

2/7/8 (Item 1 from file: 73)
DIALOG(R)File 73: EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

11344635 EMBASE No: 2001358926
Adhesion molecules and atherogenesis
Huo Y.; Ley K.
K. Ley, Department of Biomedical Engineering, University of Virginia, Health Science Center, Charlottesville, VA 22908 United States
Acta Physiologica Scandinavica (ACTA PHYSIOL. SCAND.) (United Kingdom) 2001, 173/1 (35-43)
CODEN: APSCA ISSN: 0001-6772
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

Atherosclerosis is an inflammatory disease of the vessel wall characterized by monocyte infiltration in response to pro-atherogenic factors such as oxidized lipids. Recently, the role of specific adhesion molecules in this process has been explored. The endothelium overlying **atherosclerotic** lesions expresses P-selectin and the shoulder regions express vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1), which is also expressed on endothelium in regions not prone to plaque development. Serum levels of soluble P-selectin, ICAM-1 and VCAM-1 are elevated in patients with angina pectoris or peripheral **atherosclerotic** disease. Reconstituted in vitro systems using monocytes on cytokine-activated endothelial cells under shear flow suggested the involvement of P-selectin, L-selectin, VCAM-1, its ligand, VLA-4 integrin and CD18 integrins. Studies of monocyte adhesion in isolated perfused carotid arteries harvested from **atherosclerotic** (apoE^{-/-}) mice show a predominant involvement of P-selectin and its ligand P-selectin glycoprotein-1 (PSGL-1) in rolling and of VLA-4 and VCAM-1 in firm adhesion. Consistent with these findings, apoE^{-/-} mice that are also deficient for P-selectin show significantly reduced **atherosclerotic** lesion sizes and are almost completely protected from neointimal growth after vascular injury. Milder effects are also seen in the low-density lipoprotein (LDL) receptor deficient (LDLR^{-/-}) mouse. In a high cholesterol/cholate model, a role of ICAM-1 and CD18 integrins was also shown, but this awaits confirmation in more physiologic models. Transient blockade of the VLA-4/VCAM-1 adhesion pathway by antibodies or peptides in

apoE-/- or LDLR-/- mice reduced monocyte and lipid accumulation in lesions. These data suggest that P-selectin, **PSGL-1**, VLA-4 and VCAM-1 are the most important adhesion molecules involved in monocyte recruitment to **atherosclerotic** lesions.

2/7/9 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

11332032 EMBASE No: 2001346318
High-shear-stress-induced activation of platelets and microparticles enhances expression of cell adhesion molecules in THP-1 and endothelial cells
Nomura S.; Tandon N.N.; Nakamura T.; Cone J.; Fukuhara S.; Kambayashi J.
S. Nomura, Otsuka America Pharmaceutical Inc., Rockville, MD 20850
United States
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Atherosclerosis (ATHEROSCLEROSIS) (Ireland) 2001, 158/2 (277-287)
CODEN: ATHSB ISSN: 0021-9150
PUBLISHER ITEM IDENTIFIER: S0021915001004336
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 68

Interaction between leukocyte and endothelial cells (ECs) is essential for vascular homeostasis and competent immune-inflammatory responses in vivo. Platelet-derived microparticles (PMPs) are generated by high shear stress and may appear in diseased small arteries and arterioles in various clinical settings. In this study, we used flow cytometry and confocal laser scanning microscopy to investigate the effects of high-shear-induced platelet and microparticle activation in adhesion molecules of THP-1 and ECs. We also measured the production of some cytokines and studied cytokine mRNA from THP-1 and ECs after PMP stimulation. PMP stimulation of THP-1 cells increased CD11b, CD32, and CD33 but not CD29, CD31, and CD36. PMP stimulation of ECs increased CD54 and CD63 but not CD9, CD29, and CD31. PMPs induced interleukin-8 (IL-8), interleukin-1beta (IL-1beta), and tumor necrosis factor alpha (TNFalpha) production by THP-1. PMPs also induced IL-8, IL-1beta, and interleukin-6 (IL-6) production by ECs. Production was time-dependent. With RT-PCR, some cytokine mRNAs were detected in THP-1 and ECs after PMP stimulation. In relation to adhesiveness after PMP stimulation, we could clearly observe a shift in distribution not only of CD11b in THP-1 cells but also of CD54 in ECs. In addition, anti-**P-selectin glycoprotein ligand-1** antibody reduced the expression of CD11b, CD32, and CD33 in THP-1 after PMP stimulation. These results suggest that high-shear-induced microparticles may contribute to the development of **atherosclerosis** and participate in vascular damage in inflammatory disorders. (c) 2001 Elsevier Science Ireland Ltd.

2/7/10 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE
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11302989 EMBASE No: 2001317223
Adhesive interactions of leukocytes, platelets, and the vessel wall during hemostasis and inflammation
McEver R.P.
Dr. R.P. McEver, Warren Medical Research Institute, University of Oklahoma, Health Sciences Center, 825 N. E. 13th Street, Oklahoma City, OK 73104 United States
AUTHOR EMAIL: rodger-mcever@ouhsc.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 2001, 86/3 (746-756)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal ; Review

LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 149

2/7/11 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

07741206 EMBASE No: 1999223340
P-selectin binding promotes the adhesion of monocytes to VCAM-1 under flow conditions
Yago T.; Tsukuda M.; Minami M.
Dr. M. Minami, Department of Otolaryngology, Yokohama City University, School of Medicine, Fukuura 3-9, Kanazawa-ku, Yokohama 236 Japan
Journal of Immunology (J. IMMUNOL.) (United States) 01 JUL 1999, 163/1 (367-373)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 39

This study examined the adhesive interaction of peripheral blood monocytes with VCAM-1 and analyzed the effect of P-selectin binding to monocytes on the adhesive interaction with VCAM-1 under flow conditions. **P-selectin glycoprotein ligand-1** is expressed on most monocytes. Furthermore, most monocytes bind soluble P-selectin derived from platelets. P-selectin binding to monocytes did not alter the amount of expression of alpha₄ integrin on monocytes. However, the mean channel fluorescence value for binding Cy2- conjugated soluble VCAM-1 to P-selectin-bound monocytes was slightly more than that for binding Cy2-conjugated soluble VCAM-1 to untreated monocytes. Under flow conditions, the number of P-selectin-bound monocytes bound to VCAM-1 was much higher than that of untreated monocytes bound to VCAM-1. These bindings were abolished by pretreatment of untreated monocytes and P-selectin-bound monocytes with anti-VCAM-1 mAb or anti-alpha₄ integrin mAb. Furthermore, P-selectin binding to monocytes increased shear resistance and thus increased the adhesive strength of monocytes to VCAM-1. These findings indicate that P-selectin binding to monocytes enhances the adhesive interaction of monocytes with VCAM-1. It is suggested that **P-selectin glycoprotein ligand-1/P-selectin** interaction and alpha₄ integrin/VCAM-1 interaction can act sequentially in the adhesion cascade that regulates monocyte trafficking to inflammatory and **atherosclerotic** lesion.

2/7/12 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

07381280 EMBASE No: 1998291193
Important contributions of **P-selectin glycoprotein ligand-1**-mediated secondary capture to human monocyte adhesion to P-selectin, E-selectin, and TNF-alpha-activated endothelium under flow in vitro
Lim Y.-C.; Snapp K.; Kansas G.S.; Camphausen R.; Ding H.; Luscinskas F.W.
Dr. F.W. Luscinskas, Brigham and Women's Hospital, 221 Longwood Avenue, Boston, MA 02115 United States
AUTHOR EMAIL: fluscinskas@rics.bwh.harvard.edu
Journal of Immunology (J. IMMUNOL.) (United States) 01 SEP 1998, 161/5 (2501-2508)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

In this study, an in vitro flow model and a blocking mAb to **P-**

selectin glycoprotein ligand-1 (PSGL-1) were used to define the role of **PSGL-1** in monocyte attachment and rolling on E- and P-selectin and in attachment and accumulation on 6-h TNF-alpha-activated HUVEC. KPL1, an adhesion-blocking mAb directed against the tyrosine sulfate motif of **PSGL-1**, abolished monocyte- adhesive interactions with P-selectin, but only partially blocked monocyte interaction with E-selectin. Further analysis showed that on E-selectin, KPL1 blocked only secondary (i.e., monocyte/E-selectin) interactions, but did not block primary (i.e., monocyte/E-selectin) interactions, with secondary adhesion accounting for 90% of the total adhesive interactions on either E- or P-selectin. On cytokine-activated HUVEC, monocytes initially attached and formed linear strings of adherent cells, which involved both primary and secondary adhesion. **PSGL-1** or L-selectin mAb reduced string formation, and the combination of **PSGL-1** and L-selectin mAb prevented monocyte strings and inhibited 86% of accumulation. Monocyte attachment and rolling on purified adherent monocytes were also critically dependent on **PSGL-1** on the adherent monocytes. These studies document that secondary interactions between monocytes, mediated by **PSGL-1**, are crucial for monocyte initial attachment, rolling, and accumulation on activated endothelium under laminar shear flow.

2/7/13 (Item 1 from file: 399)
 DIALOG(R)File 399:CA SEARCH(R)
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133280563 CA: 133(20)280563a PATENT
 Human antibodies that bind human IL-12 and methods for producing
 INVENTOR(AUTHOR): Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael;
 Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra;
 Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela;
 Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela;
 Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara;
 Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
 LOCATION: Germany,
 ASSIGNEE: Basf A.-G.; Genetics Institute Inc.; et al.
 PATENT: PCT International ; WO 200056772 A1 DATE: 20000928
 APPLICATION: WO 2000US7946 (20000324) *US PV126603 (19990325)
 PAGES: 377 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07K-016/24A;
 C12N-015/13B; C12N-015/63B; C12N-005/10B; C07K-016/00B; A61K-039/395B;
 G01N-033/577B; C12P-021/08B; A61P-043/00B DESIGNATED COUNTRIES: AE; AG; AL
 ; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM;
 DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP;
 KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL;
 PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN;
 YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM
 ; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR;
 GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML;
 MR; NE; SN; TD; TG
 SECTION:
 CA215003 Immunochemistry
 CA203XXX Biochemical Genetics
 IDENTIFIERS: human antibody interleukin 12 autoimmune disease,
 inflammation recombinant antibody human interleukin 12
 DESCRIPTORS:
 Immunoglobulins...
 A; recombinant human antibodies that bind human IL-12 for treatment of
 autoimmune diseases and inflammatory diseases
 Respiratory distress syndrome...
 adult; recombinant human antibodies that bind human IL-12 for treatment
 of autoimmune diseases and inflammatory diseases
 Interleukin 2 receptors...
 .alpha.-chain; recombinant human antibodies that bind human IL-12 for
 treatment of autoimmune diseases and inflammatory diseases
 Spinal column...
 ankylosing spondylitis; recombinant human antibodies that bind human

IL-12 for treatment of autoimmune diseases and inflammatory diseases

- Transforming growth factors...
 - .beta.-; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Interferons...
 - .beta.1, .beta.1a and .beta.1b; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Phytohemagglutinins...
 - blast proliferation assay; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Drug delivery systems...
 - carriers; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Antigens...
 - CD90; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Fatigue,biological...
 - chronic fatigue syndrome; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunoglobulins...
 - conjugates; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Intestine,disease...
 - Crohn's; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Anti-inflammatory agents...
 - cytokine; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunity...
 - disorder, acute and chronic; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Blood coagulation...
 - disseminated intravascular; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunoglobulins...
 - E; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Cytokines...
 - EMAP-II or endothelial-monocyte-activating polypeptide II; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Heart,disease...
 - failure; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Lung,disease...
 - fibrosis; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunoglobulins...
 - fragments; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Transplant and Transplantation...
 - graft-vs.-host reaction; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunoglobulins...
 - G1; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunoglobulins...
 - G2; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunoglobulins...
 - G3; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
- Immunoglobulins...

G4; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Immunoglobulins...
heavy chains; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Anemia(disease)...
hemolytic; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Purpura(disease)...
Henoch-Schoenlein's; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Nervous system...
Huntington's chorea; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Tumor necrosis factor receptors...
Ig conjugates; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Heart,disease...
infarction; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Parasite...
infection; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Cytokines...
inflammatory, anti-; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Intestine,disease...
inflammatory; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Complement... Signal transduction,biological... Thromboxanes...
inhibitors; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Drug delivery systems...
injections, i.v.; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Diabetes mellitus...
insulin-dependent; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Baboon... Chimpanzee... Macaca irus... Macaca mulatta... Marmoset...

Primate...
interleukin 12; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Rheumatoid arthritis...
juvenile; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Blood vessel,disease...
Kawasaki; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Immunoglobulins...
light chains; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Immunoglobulins...
M; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Antibodies...
monoclonal; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Spinal cord...
myelitis, acute transverse; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Kidney,disease...
nephrotic syndrome; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Antibodies...
neutralizing; recombinant human antibodies that bind human IL-12 for

treatment of autoimmune diseases and inflammatory diseases

Anti-inflammatory agents...

nonsteroidal; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Selectins...

P-, glycoprotein ligand; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Glycoproteins, specific or class...

p-selectin glycoprotein ligand; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Artery, disease...

periarteritis nodosa; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Bioassay...

phytohemagglutinin blast proliferation assay; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Biliary tract...

primary biliary cirrhosis; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

Arthritis...

psoriatic arthritis; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

(etc.)...

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297777-31-2 297777-32-3 297777-33-4 297777-34-5 297777-35-6 amino acid sequence; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

28088-64-4D analogs, recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

58-61-7 biological studies, agonists; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

7782-44-7 biological studies, hyperbaric; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

110-86-1D imidazole compds., recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

80449-02-1 142243-02-5 inhibitor; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

9004-06-2 9015-82-1 9025-82-5 9029-60-1 122191-40-6 151769-16-3 inhibitors; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

9036-21-9 IV, inhibitor; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

288-32-4D pyridinyl compds., recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

50-02-2 50-18-0 50-24-8 50-44-2 59-05-2 83-43-2 89-57-6 443-48-1

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297738-67-1	297738-68-2	297738-69-3	297738-70-6	297738-71-7
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297738-82-0	297738-83-1	297738-84-2	297738-85-3	297738-86-4
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297738-92-2	297738-93-3	297738-94-4	297738-95-5	297738-96-6
297738-97-7	297738-98-8	297738-99-9	297739-00-5	297739-01-6
297739-02-7	297739-03-8	297739-04-9	297739-05-0	297739-06-1
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297739-12-9	297739-13-0	297739-14-1	297739-15-2	297739-16-3
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297740-67-1	297740-68-2	298200-40-5	298200-41-6	298200-42-7
298200-43-8	298200-44-9	298200-45-0	298200-46-1	298200-47-2
298200-48-3	298200-49-4	298200-50-7	recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases	
226983-90-0	226984-98-1	297782-45-7	297782-46-8	297782-47-9
297782-48-0	297782-49-1	297782-50-4	297782-51-5	unclaimed nucleotide sequence; human antibodies that bind human IL-12 and methods for producing
260430-73-7	297782-22-0	297782-23-1	297782-24-2	297782-25-3
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 297782-36-6 297782-37-7 297782-38-8 297782-39-9 297782-40-2
 297782-41-3 297782-42-4 297782-43-5 297782-44-6 297782-52-6
 297782-53-7 298200-51-8 298200-52-9 298200-53-0 unclaimed protein
 sequence; human antibodies that bind human IL-12 and methods for
 producing
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 297737-56-5 297737-57-6 297737-58-7 297737-59-8 297737-60-1
 297737-61-2 297737-62-3 297737-63-4 297737-64-5 297737-65-6
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 297737-71-4 297737-72-5 297737-73-6 297737-74-7 297737-75-8
 297737-76-9 297737-77-0 297737-78-1 297737-79-2 297737-80-5
 297737-81-6 297737-82-7 297737-83-8 297737-84-9 297737-85-0
 297737-86-1 297737-87-2 297737-88-3 297737-89-4 297737-90-7
 297737-91-8 297737-92-9 297737-93-0 297737-94-1 297737-95-2
 297737-96-3 297737-97-4 297737-98-5 297737-99-6 297738-00-2
 297738-01-3 297738-02-4 297738-03-5 297738-04-6 unclaimed sequence;
 human antibodies that bind human IL-12 and methods for producing

2/7/14 (Item 2 from file: 399)
 DIALOG(R)File 399:CA SEARCH(R)
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124135703 CA: 124(11)135703f PATENT
 Method using agents inhibiting interaction between P-selectin??? and a
 P-selectin ligand for treating and preventing atherosclerosis
 INVENTOR(AUTHOR): Wagner, Denisa D.; Johnson, Robert C.
 LOCATION: USA
 ASSIGNEE: Center for Blood Research, Inc.
 PATENT: PCT International ; WO 9533484 A1 DATE: 951214
 APPLICATION: WO 95US6940 (950601) *US 253663 (940603) *US 377798 (950124)
 PAGES: 35 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A;
 A61K-038/02B; A61K-038/16B; A61K-031/70B DESIGNATED COUNTRIES: CA; JP
 DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC;
 NL; PT; SE
 SECTION:
 CA201008 Pharmacology
 IDENTIFIERS: P selectin ligand inhibition atherosclerosis treatment
 DESCRIPTORS:
 Plant...

agents derived from plant ext. for inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Heart,disease, restenosis...
agents for inhibiting interaction between P-selectin and P-selectin
ligand for treating and preventing atherosclerosis
Blood platelet... Eosinophil... Leukocyte... Lymphocyte,natural killer cell
... Monocyte... Neutrophil...
agents inhibiting interaction between cellular P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Antiarteriosclerotics,antiatherosclerotics... Blood-group substances,Lea,
sialyl... Blood-group substances,Lex, sialyl... Carbohydrates and
Sugars,biological studies... Glycoproteins,biological studies...
Glycoproteins,specific or class, PSGL-1 (P-selectin glycoprotein ligand-1)
... Ligands... Receptors,P-selectins...
agents inhibiting interaction between P-selectin and P-selectin ligand
for treating and preventing atherosclerosis
Lymphocyte,T-cell...
CD4+ and CD8+; agents inhibiting interaction between cellular
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Artery,endothelium...
cell; agents inhibiting interaction between cellular P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Antibodies... Antibodies,monoclonal... Peptides,biological studies...
Proteins,biological studies... Sulfatides...
inhibitory; agents inhibiting interaction between P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Lysosome...
membrane, glycoproteins; agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Ligands...
P-selectin, 160 kD monospecific; agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Blood-group substances,Lex, sialyl...
pentasaccharide; agents inhibiting interaction between P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Mucopolysaccharides,lactosaminoglycans,biological studies...
poly-; agents inhibiting interaction between P-selectin and P-selectin
ligand for treating and preventing atherosclerosis
Venoms...
snake; agents derived from snake venom for inhibiting interaction
between P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Snake...
venom; agents derived from snake venom for inhibiting interaction
between P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Carbohydrates and Sugars,biological studies...
2,6-sialic acid-contg.; agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Blood-group substances,Lex...
3'-O-sulfate; agents inhibiting interaction between P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
CAS REGISTRY NUMBERS:
9005-49-6D oligosaccharides, agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
? s (psgl? or p(w)selectin(w)glycoprotein(w)ligand) and (restenosis)

746 PSGL?
3756683 P
24823 SELECTIN

250085 GLYCOPROTEIN
293792 LIGAND
739 P(W) SELECTIN(W) GLYCOPROTEIN(W) LIGAND
24623 RESTENOSIS
S3 13 (PSGL? OR P(W) SELECTIN(W) GLYCOPROTEIN(W) LIGAND) AND
(RESTENOSIS)

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...completed examining records

S4 9 RD S3 (unique items)

? t s4/7/all

4/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS Previews(R)
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13205270 BIOSIS NO.: 200100412419
Prevention of intimal hyperplasia with recombinant soluble **P-selectin glycoprotein ligand**-immunoglobulin in the porcine coronary artery balloon injury model.
AUTHOR: Wang Kai; Zhou Zhongmin; Zhou Xiaorong; Tarakji Khaldoun; Topol Eric J; Lincoff A Michael(a)
AUTHOR ADDRESS: (a)Cardiology Department, Cleveland Clinic Foundation, 9500 Euclid Ave., F25, Cleveland, OH, 44195: lincofa@ccf.org**USA
JOURNAL: Journal of the American College of Cardiology 38 (2):p577-582
August, 2001
MEDIUM: print
ISSN: 0735-1097
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: OBJECTIVES: The role of P-selectin in the process of **restenosis** was evaluated using a recombinant immunoglobulin (Ig) chimera form of its ligand, soluble **P-selectin glycoprotein ligand**-Ig (rPSGL-Ig), as a competitive inhibitor for the natural ligand on leukocytes. BACKGROUND: Inflammation and coagulation activation after vascular injury may be an important factor in the development of **restenosis**. P-selectin has been shown to mediate leukocyte-endothelium and leukocyte-platelet interaction. These interactions are mediated through binding of P-selectin to **P-selectin glycoprotein ligand-1 (PSGL-1)** located on the surface of leukocytes. METHODS: Balloon injury was induced in the left anterior descending and right coronary arteries of 16 pigs at a balloon/artery diameter ratio of 1.5:1. Either rPSGL-Ig (1 mg/kg) or saline was randomly administered 15 min before balloon injury as an intravenous bolus. Four weeks after injury, morphometric analysis, immunohistochemistry and histological evaluation were performed on injured arterial segments. RESULTS: Increased luminal area was found in the rPSGL-Ig group compared with the placebo group (1.63 \pm 0.57 mm² vs. 1.26 \pm 0.32 mm², p=0.044) owing to significantly reduced neointimal hyperplasia (cross-sectional area, 0.46 \pm 0.45 mm² vs. 0.13 \pm 0.11 mm², p=0.013). Immunohistochemistry and histological evaluation showed a significant decrease in the presence of tumor necrosis factor-alpha, interleukin-1 beta, and infiltration of macrophages in the injured vessel segments in the rPSGL-Ig group. CONCLUSIONS: P-selectin antagonism using rPSGL-Ig decreases neointimal hyperplasia following balloon injury, by inhibiting the inflammatory and thrombotic responses at the site of balloon injury, which appears to play a pivotal role in the pathogenesis of **restenosis**.

4/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS Previews(R)

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13115159 BIOSIS NO.: 200100322308

Alterations in platelet thrombus formation, leukocyte recruitment, and intimal hyperplasia in P-selectin-deficient mice after transluminal femoral artery injury.

AUTHOR: Smyth Susan S(a); Reis Ernane D; Fallon John T; Gordon Ron; Collier Barry S(a)

AUTHOR ADDRESS: (a)Medicine, Mount Sinai School of Medicine, New York, NY** USA

JOURNAL: Blood 96 (11 Part 1):p813a November 16, 2000

MEDIUM: print

CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000

SPONSOR: American Society of Hematology

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: Platelet activation at the site of vascular injury, such as occurs after atherosclerotic plaque rupture or following percutaneous intervention, results in platelet-neutrophil interactions, which contribute to local thrombosis, downstream microcirculatory events, and systemic inflammation. The initial interaction of activated platelets with neutrophils is mediated by platelet P-selectin binding to neutrophil **PSGL-1**. To investigate the role of platelet-neutrophil interactions in response to arterial injury, we performed transluminal, wire-induced injury to the femoral artery of wild-type C57Bl/6 mice (n=26) and P-selectin -/- mice (n=26). The femoral arteries of anesthetized male mice aged 8-10 weeks were injured by passing a 0.25 mm wire in the lumen of the femoral artery three times, and the mice were euthanized by perfusion fixation 1 h or 4 weeks after injury. 1 h after injury in wild-type mice, platelets were found adherent to the blood vessel wall and neutrophils were attached to the platelets. As viewed by TEM and SEM, the platelet layer varied between 1 and approx 3 platelets thick, and many of the platelets in contact with the wall were spread and at least partially degranulated. 1 h after injury of P-selectin -/- mice, the platelet layer appeared less compact and appeared to extend further into the lumen; moreover, more of the platelets appeared to retain their granules. There was a striking decrease in leukocyte attachment to the platelets. Four weeks after injury, the neointimal area in P-selectin -/- mice (2,100 +/- 900 μm^2) was significantly smaller than in the wild-type mice (10,200 +/- 2,100 μm^2) (p=0.004). These results indicate that P-selectin is required for neutrophil recruitment to platelets lining the vessel wall 1 h after injury and P-selectin deficiency protects mice from developing intimal hyperplasia 4 weeks after injury. Moreover, in the P-selectin -/- mice, the platelets depositing on the damaged vessel appeared to be less activated and to extend further into the lumen, suggesting that P-selectin may play a role in platelet activation and platelet thrombus formation. Our data support the possibility that antagonists to P-selectin may decrease intimal hyperplasia and clinical **restenosis** after percutaneous vascular interventions in humans.

4/7/3 (Item 3 from file: 5)
DIALOG(R)File 5: Biosis Previews(R)
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12954079 BIOSIS NO.: 200100161228

Recombinant soluble **P-selectin glycoprotein ligand**

-1-Ig reduces **restenosis** through inhibition of platelet-neutrophil adhesion after double angioplasty in swine.

AUTHOR: Bienvenu Jean-Guy; Tanguay Jean-Francois; Theoret Jean-Francois; Kumar Anjali; Schaub Robert G; Merhi Yahye(a)

AUTHOR ADDRESS: (a)Laboratory of Experimental Pathology, Montreal Heart

Institute, 5000 Belanger St E, Montreal, PQ, H1T 1C8:
merhi@icm.umontreal.ca**Canada
JOURNAL: Circulation 103 (8):p1128-1134 February 27, 2001
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Background: P-selectin mediates leukocyte recruitment to activated platelets and endothelium through its high-affinity receptor **P-selectin glycoprotein ligand-1 (PSGL-1)**. Platelet and leukocyte activation and binding have been reported after coronary angioplasty and were correlated with **restenosis**. We investigated the effect of a recombinant soluble **PSGL-1 (rPSGL-Ig)** on the adhesion of platelets and neutrophils and the development of **restenosis** after double arterial injury. Methods and Results: Four weeks after angioplasty of both carotid arteries in pigs, a second angioplasty was performed at the same sites, 15 minutes after a single administration of vehicle or rPSGL-1 (1 mg/kg IV). Animals were euthanized 1 hour, 4 hours, 1 week, or 4 weeks later. Adhesion of autologous 51Cr-platelets and 111In-neutrophils was quantified and histological/morphometric analyses were performed. Although rPSGL-Ig did not affect adherence of these cells 1 hour after injury, it significantly reduced the adhesion of platelets (50% at 4 hours and 85% at 1 week) and neutrophils (50% at 4 hours and 78% at 1 week) to deeply injured arteries. At 4 weeks, the residual lumen was 63% larger in rPSGL-Ig-treated arteries as compared with control arteries (6.1 ± 0.6 versus 3.8 ± 0.1 mm²; $P < 0.002$). The neointimal area was slightly reduced (0.5 in rPSGL-Ig versus 0.7 mm² in control). The ratio of the external elastic lamina of injured to uninjured reference segments was >1 in treated arteries and <1 in control arteries. Conclusions: P-selectin antagonism with rPSGL-Ig inhibits early platelet/leukocyte adhesion on injured arteries and reduces **restenosis** through a positive impact on vascular remodeling. Hence, rPSGL-Ig may have potential in the prevention of **restenosis**.

4/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12953911 BIOSIS NO.: 200100161060
rPSGL-Ig inhibits in-stent **restenosis** in porcine coronary arteries after double injury.
AUTHOR: Tanguay Jean-Francois(a); Geoffroy Pascale; Theoret Jean-Francois; Schaub Robert G; Kumar Anjali; Merhi Yahye
AUTHOR ADDRESS: (a)Montreal Heart Institute, Montreal, PQ**Canada
JOURNAL: Journal of the American College of Cardiology 37 (2 Supplement A):p28A February, 2001
MEDIUM: print
CONFERENCE/MEETING: 50th Annual Scientific Session of the American College of Cardiology Orlando, Florida, USA March 18-21, 2001
ISSN: 0735-1097
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12905204 BIOSIS NO.: 200100112353
Prevention of intimal hyperplasia with recombinant soluble P-

selectin glycoprotein ligand-Ig in the porcine coronary artery balloon injury model.
AUTHOR: Wang Kai(a); Zhou Zhong Min(a); Zhou Xiaorong(a); Lincoff A Michael (a)
AUTHOR ADDRESS: (a)Cleveland Clin Fdn, Cleveland, OH**USA
JOURNAL: Circulation 102 (18 Supplement):pII329-II330 October 31, 2000
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

12839849 BIOSIS NO.: 200100046998
Effect of a recombinant soluble **P-selectin glycoprotein ligand-1** on **restenosis** following arterial injury by repeat angioplasty in pigs.
AUTHOR: Bienvenu J G(a); Tanguay J F; Theoret J F; Kumar A; Schaub R G; Merhi Y
AUTHOR ADDRESS: (a)Montreal, PQ**Canada
JOURNAL: Canadian Journal of Cardiology 16 (Supplement F):p213F-214F September, 2000
MEDIUM: print
CONFERENCE/MEETING: 53rd Annual Meeting of the Canadian Cardiovascular Society Vancouver, British Columbia, Canada October 20-November 01, 2000
SPONSOR: Canadian Cardiovascular Society
ISSN: 0828-282X
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12411198 BIOSIS NO.: 200000164700
Effect of a recombinant soluble **P-Selectin Glycoprotein Ligand-1** chimera on **restenosis** following arterial injury by repeat angioplasty in pigs.
AUTHOR: Bienvenu Jean-Guy(a); Tanguay Jean-Francois; Theoret Jean-Francois; Kumar Anjali; Schaub Robert G; Merhi Yahye
AUTHOR ADDRESS: (a)Montreal Heart Institute, Montreal, PQ**Canada
JOURNAL: Journal of the American College of Cardiology. 35 (2 suppl. A):p 16A Feb., 2000
CONFERENCE/MEETING: 29th Annual Scientific Session of the American College of Cardiology. Anaheim, California, USA March 12-15, 2000
ISSN: 0735-1097
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/8 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

07629695 EMBASE No: 1999099739
Recombinant soluble form of **PSGL-1** accelerates thrombolysis and prevents reocclusion in a porcine model

Kumar A.; Villani M.P.; Patel U.K.; Keith J.C. Jr.; Schaub R.G.
Dr. A. Kumar, Preclinical R and D, Genetics Institute, Inc., One Burt
Rd, Andover, MA 01810 United States
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Circulation (CIRCULATION) (United States) 16 MAR 1999, 99/10
(1363-1369)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 42

Background - We investigated whether administration of a soluble recombinant **P-selectin glycoprotein ligand-1** chimera (rPSGL-Ig) in conjunction with thrombolytic therapy would enhance thrombolysis by preventing ongoing interactions of leukocytes with platelets and the injured arterial wall. Methods and Results - An occlusive thrombus was formed in an internal iliac artery of Yorkshire pigs by placement of a copper coil in the artery under fluoroscopic guidance. Pigs then received heparin and, 15 minutes later, either vehicle or rPSGL-Ig followed by infusion with 25 mg tissue plasminogen activator according to the 90-minute regimen. Blood flow through the artery was monitored by angiography and scored on a scale of 0 to 3. Lysis of the thrombus was accelerated by 70% in pigs treated with rPSGL-Ig 250 mug/kg compared with control (13.3+/-5.0 versus 44.4+/-13.3 minutes; n=9 each). Eight of 9 control pigs reoccluded in 13.8+/-16.9 minutes after the end of tissue plasminogen activator infusion, whereas no reocclusion was observed in 8 of 9 pigs in the rPSGL-Ig group. When the dose of rPSGL-Ig was increased to 500 mug/kg, time to lysis was shortened by 61% from control (18.0+/-8.4 versus 46.0+/-8.9 minutes). Reocclusion occurred in 6.0+/-15.2 minutes in control but not in any rPSGL-Ig-treated pig (n=5 each). In addition, near-normal flow (score 2 or 3) after thrombolysis was achieved 59% and 58% faster in the 2 rPSGL-Ig groups than in their respective controls. Conclusions - Inhibition of leukocyte accumulation at the site of thrombosis with rPSGL-Ig may represent a safe therapeutic intervention that could be important in accelerating thrombolysis, achieving optimal reperfusion, and reducing incidence of acute reocclusion.

4/7/9 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

124135703 CA: 124(11)135703f PATENT
Method using agents inhibiting interaction between P-selectin??? and a P-selectin ligand for treating and preventing atherosclerosis
INVENTOR(AUTHOR): Wagner, Denisa D.; Johnson, Robert C.
LOCATION: USA
ASSIGNEE: Center for Blood Research, Inc.
PATENT: PCT International ; WO 9533484 A1 DATE: 951214
APPLICATION: WO 95US6940 (950601) *US 253663 (940603) *US 377798 (950124)
PAGES: 35 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A;
A61K-038/02B; A61K-038/16B; A61K-031/70B DESIGNATED COUNTRIES: CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC;
NL; PT; SE
SECTION:
CA201008 Pharmacology
IDENTIFIERS: P selectin ligand inhibition atherosclerosis treatment
DESCRIPTORS:
Plant...
agents derived from plant ext. for inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Heart,disease, restenosis...
agents for inhibiting interaction between P-selectin and P-selectin
ligand for treating and preventing atherosclerosis
Blood platelet... Eosinophil... Leukocyte... Lymphocyte,natural killer cell

... Monocyte... Neutrophil...
agents inhibiting interaction between cellular P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Antiartherosclerotics, antiatherosclerotics... Blood-group substances, Lea,
sialyl... Blood-group substances, Lex, sialyl... Carbohydrates and
Sugars, biological studies... Glycoproteins, biological studies...
Glycoproteins, specific or class, PSGL-1 (P-selectin glycoprotein ligand-1)
... Ligands... Receptors, P-selectins...
agents inhibiting interaction between P-selectin and P-selectin ligand
for treating and preventing atherosclerosis
Lymphocyte, T-cell...
CD4+ and CD8+; agents inhibiting interaction between cellular
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Artery, endothelium...
cell; agents inhibiting interaction between cellular P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Antibodies... Antibodies, monoclonal... Peptides, biological studies...
Proteins, biological studies... Sulfatides...
inhibitory; agents inhibiting interaction between P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Lysosome...
membrane, glycoproteins; agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Ligands...
P-selectin, 160 kD monospecific; agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Blood-group substances, Lex, sialyl...
pentasaccharide; agents inhibiting interaction between P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
Mucopolysaccharides, lactosaminoglycans, biological studies...
poly-; agents inhibiting interaction between P-selectin and P-selectin
ligand for treating and preventing atherosclerosis
Venoms...
snake; agents derived from snake venom for inhibiting interaction
between P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Snake...
venom; agents derived from snake venom for inhibiting interaction
between P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Carbohydrates and Sugars, biological studies...
2,6-sialic acid-contg.; agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
Blood-group substances, Lex...
3'-O-sulfate; agents inhibiting interaction between P-selectin and
P-selectin ligand for treating and preventing atherosclerosis
CAS REGISTRY NUMBERS:
9005-49-6D oligosaccharides, agents inhibiting interaction between
P-selectin and P-selectin ligand for treating and preventing
atherosclerosis
? ds

Set	Items	Description
S1	25	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHER- OSCLER?)
S2	14	RD S1 (unique items)
S3	13	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTE- NOSIS)
S4	9	RD S3 (unique items)

? s s2 and s4

Set	Items	Description
S1	25	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHER- OSCLER?)
S2	14	RD S1 (unique items)
S3	13	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTE- NOSIS)
S4	9	RD S3 (unique items)
S5	2	S2 AND S4
S6	2	RD S5 (unique items)
S7	3360	RESTENOSIS AND ATHEROSCLEROSIS
S8	495	S7 AND REVIEW?
S9	303	RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10	0	S9 AND P(W)SELECTION?
S11	1	S9 AND P(W)SELECTIN?

? s (restenosis or atherosclerosis) and p(w)selectin?

24623 RESTENOSIS
136871 ATHEROSCLEROSIS
3756683 P
66839 SELECTIN?
9434 P(W)SELECTIN?
S12 511 (RESTENOSIS OR ATHEROSCLEROSIS) AND P(W)SELECTIN?
? s s12 and review?

511 S12
3019137 REVIEW?
S13 27 S12 AND REVIEW?
? rd s13

...completed examining records
S14 21 RD S13 (unique items)
? t s14/3/all

14/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

13218531 BIOSIS NO.: 200100425680
Early increase in levels of soluble inter-cellular adhesion molecule-1
(sICAM-1): Potential risk factor for the acute coronary syndromes.
AUTHOR: O'Malley T; Ludlam C A; Riemersma R A; Fox K A A(a)
AUTHOR ADDRESS: (a)Cardiovascular Research, Department of Medical and
Radiological Sciences, Cardiology, The University of Edinburgh, Royal
Infirmary of Edinburgh, Lauriston Place, Edinburgh, EH3 9YW**UK
JOURNAL: European Heart Journal 22 (14):p1226-1234 July, 2001
MEDIUM: print
ISSN: 0195-668X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

14/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

11352656 BIOSIS NO.: 199800133988
Platelet-leukocyte-cross-talk in diabetes mellitus.
AUTHOR: Tschoepe D(a); Rauch U; Schwippert B
AUTHOR ADDRESS: (a)Diabetes Research Inst. at Henrich Heine Univ., Cellular
Haemostasis and Clinical Angiology Group**Germany
JOURNAL: Hormone and Metabolic Research 29 (12):p631-635 Dec., 1997
ISSN: 0018-5043
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

14/3/3 (Item 3 from file: 5)
DIALOG(R)File 5: Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09050005 BIOSIS NO.: 199497058375
Potential roles for oxidized phospholipids in inflammation and
atherogenesis.
BOOK TITLE: **Atherosclerosis Reviews; Atherosclerosis:**
Cellular interactions, growth factors, and lipids
AUTHOR: Prescott Stephen M(a); Patel Kamala D; Smiley Patricia L;
Stafforini Diana M; Lorant Diane E; Zimmerman Guy A; McIntyre Thomas M
BOOK AUTHOR/EDITOR: Weber P C; Leaf A: Eds
AUTHOR ADDRESS: (a)Eccles Inst. Human Genet., Room 4220, Univ. Utah, Salt
Lake City, UT 84112**USA
JOURNAL: Atherosclerosis Reviews 25p59-68 1993
BOOK PUBLISHER: Raven Press, 1185 Avenue of the Americas, New York, New
York 10036-2806, USA
CONFERENCE/MEETING: Third Miles International Workshop on Atherosclerosis
Stresa, Italy September 30-October 2, 1992
ISSN: 0362-1650 ISBN: 0-7817-0099-X
RECORD TYPE: Citation
LANGUAGE: English

14/3/4 (Item 1 from file: 73)
DIALOG(R)File 73: EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

11358152 EMBASE No: 2001372385
The vascular-associated lymphoid tissue: A new site of local immunity
Millonig G.; Schwentner C.; Mueller P.; Mayerl C.; Wick G.
Dr. G. Wick, Inst. for Biomedical Aging Research, Austrian Academy of
Sciences, Rennweg 10, 6020 Innsbruck Austria
AUTHOR EMAIL: georg.wick@uibk.ac.at
Current Opinion in Lipidology (CURR. OPIN. LIPIDOLOGY) (United Kingdom)
2001, 12/5 (547-553)
CODEN: COPLE ISSN: 0957-9672
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 48

14/3/5 (Item 2 from file: 73)
DIALOG(R)File 73: EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

11344635 EMBASE No: 2001358926
Adhesion molecules and atherogenesis
Huo Y.; Ley K.
K. Ley, Department of Biomedical Engineering, University of Virginia,
Health Science Center, Charlottesville, VA 22908 United States
Acta Physiologica Scandinavica (ACTA PHYSIOL. SCAND.) (United Kingdom)
2001, 173/1 (35-43)
CODEN: APSCA ISSN: 0001-6772

DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

14/3/6 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

11302989 EMBASE No: 2001317223
Adhesive interactions of leukocytes, platelets, and the vessel wall
during hemostasis and inflammation
McEver R.P.
Dr. R.P. McEver, Warren Medical Research Institute, University of
Oklahoma, Health Sciences Center, 825 N. E. 13th Street, Oklahoma City,
OK 73104 United States
AUTHOR EMAIL: rodger-mcever@ouhsc.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 2001, 86/3
(746-756)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 149

14/3/7 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

10888487 EMBASE No: 2000349489
Inhibition of cellular action of thrombin by N3-cyclopropyl-7-[[4-(1-
methylethyl)phenyl]methyl]-7H-pyrrolo[3,2-f]quinazoline-1,3-diamine (SCH
79797), a nonpeptide thrombin receptor antagonist
Ahn H.-S.; Foster C.; Boykow G.; Stamford A.; Manna M.; Graziano M.
Dr. H.-S. Ahn, Department of CNS, CV Biological Research, Schering-Plough
Research Institute, 2015 Galloping Hill Road, Kenilworth, NJ 07033-1300
United States
AUTHOR EMAIL: ho-sam.ahn@spcorp.com
Biochemical Pharmacology (BIOCHEM. PHARMACOL.) (United States) 15 NOV
2000, 60/10 (1425-1434)
CODEN: BCPCA ISSN: 0006-2952
PUBLISHER ITEM IDENTIFIER: S0006295200004603
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 52

14/3/8 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

10863398 EMBASE No: 2000345938
Roles of **P-selectin** in inflammation, neointimal formation,
and vascular remodeling in balloon-injured rat carotid arteries
Hayashi S.-I.; Watanabe N.; Nakazawa K.; Suzuki J.; Tsushima K.; Tamatani
T.; Sakamoto S.; Isobe M.
Dr. M. Isobe, Dept. of Cardiovascular Medicine, Tokyo Medical and Dental
University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519 Japan
AUTHOR EMAIL: isobemi.med3@med.tmd.ac.jp
Circulation (CIRCULATION) (United States) 03 OCT 2000, 102/14
(1710-1717)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24

14/3/9 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

07291663 EMBASE No: 1998185085
Effects of viral activation of the vessel wall on inflammation and thrombosis
Vercellotti G.M.
Prof. G.M. Vercellotti, Univ. of Minnesota Medical School, Box 293 Mayo, 420 Delaware St SE, Minneapolis, MN 55105 United States
Blood Coagulation and Fibrinolysis (BLOOD COAGUL. FIBRINOLYSIS) (United Kingdom) 1998, 9/SUPPL.. 2 (S3-S6)
CODEN: BLFIE ISSN: 0957-5235
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24

14/3/10 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

07050505 EMBASE No: 1997332349
Thrombosis and **atherosclerosis**
Holvoet P.; Collen D.
P. Holvoet, Center Molecular Vascular Biology, University of Leuven, Campus Gasthuisberg, Herestraat 49, B-3000 Leuven Belgium
Current Opinion in Lipidology (CURR. OPIN. LIPIDOLOGY) (United Kingdom) 1997, 8/5 (320-328)
CODEN: COPLE ISSN: 0957-9672
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 89

14/3/11 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

06849256 EMBASE No: 1997131844
Microcirculation, vitamin E and omega 3 fatty acids: An overview
Bruckner G.
G. Bruckner, Division Clinical Nutrition, Department of Clinical Sciences, University of Kentucky, Lexington, KY 40506-0080 United States
Advances in Experimental Medicine and Biology (ADV. EXP. MED. BIOL.) (United States) 1997, 415/- (195-208)
CODEN: AEMBA ISSN: 0065-2598
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 104

14/3/12 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

06492983 EMBASE No: 1996159307
Inflammation as an early component of **atherosclerosis** and vascular damage: A role for **P-selectin** and platelet-activating factor
Prescott S.M.; McIntyre T.M.; Zimmerman G.A.; Stafforini D.M.
Program in Human Molecular Biology, Genetics Eccles Human Genetics Inst., University of Utah, Salt Lake City, UT 84112 United States
Japanese Circulation Journal (JPN. CIRC. J.) (Japan) 1996, 60/3 (137-141)
CODEN: JCIRA ISSN: 0047-1828

DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH

14/3/13 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

05383475 EMBASE No: 1993151574
Blood monocyte adhesion to vascular endothelial cells. Implication in
vascular pathology
Dosquet C.; Wautier J.-L.
Lab de Biologie Vasculaire/, Cellulaire, Hopital Lariboisiere, 2 rue
Ambroise Pare, 75010 Paris France
Clinical Hemorheology (CLIN. HEMORHEOL.) (United States) 1992, 12/6
(817-829)
CODEN: CLHED ISSN: 0271-5198
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

14/3/14 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

05336497 EMBASE No: 1993104582
Platelet alpha-granules
Harrison P.; Cramer E.M.
Coagulation Research, Rayne Institute St, Thomas' Hospital, London SE1 7EH
United Kingdom
Blood Reviews (BLOOD REV.) (United Kingdom) 1993, 7/1 (52-62)
CODEN: BLORE ISSN: 0268-960X
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

14/3/15 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11750624 21379944 PMID: 11487452
Platelet-endothelial interactions in **atherosclerosis**.
Sachais BS
Department of Pathology and Laboratory Medicine, University of
Pennsylvania, 3620 Hamilton Walk, 207A John Morgan Building, Philadelphia,
PA 19104, USA. sachais@mail.med.upenn.edu
Current atherosclerosis reports (United States) Sep 2001, 3 (5)
p412-6, ISSN 1523-3804 Journal Code: DYL
Languages: ENGLISH
Document type: Journal Article; Review; Review, Tutorial
Record type: Completed

14/3/16 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

130151597 CA: 130(12)151597d JOURNAL
Tissue factor expression by monocytes: regulation and pathophysiological
roles
AUTHOR(S): Osterud, B.
LOCATION: Department of Biochemistry, Institute of Medical Biology,
University of Tromso, Tromso, Norway
JOURNAL: Blood Coagulation Fibrinolysis DATE: 1998 VOLUME: 9 NUMBER:
Suppl. 1 PAGES: S9-S14 CODEN: BLFIE7 ISSN: 0957-5235 LANGUAGE: English
PUBLISHER: Lippincott-Raven Publishers

14/3/17 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

129120808 CA: 129(10)120808y JOURNAL
Atherosclerosis and activation of platelet and blood coagulation
AUTHOR(S): Komiyama, Yutaka; Takahashi, Hakuo
LOCATION: Dep. Clin. Sci. Lab. Med., Kansai Med. Univ., Moriguchi, Japan,
570-8507
JOURNAL: Rinsho Byori DATE: 1998 VOLUME: 46 NUMBER: 7 PAGES: 678-683
CODEN: RBYOAI ISSN: 0047-1860 LANGUAGE: Japanese PUBLISHER: Rinsho
Byori Kankokai

14/3/18 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

129079783 CA: 129(7)79783w JOURNAL
Endothelial adhesion molecules in health and disease
AUTHOR(S): Cotran, R. S.; Mayadas-Norton, T.
LOCATION: Department of Pathology, Harvard Medical School, Boston, MA,
02115, USA
JOURNAL: Pathol. Biol. DATE: 1998 VOLUME: 46 NUMBER: 3 PAGES: 164-170
CODEN: PTBIAN ISSN: 0031-3009 LANGUAGE: English PUBLISHER: Expansion
Scientifique Publications

14/3/19 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

128113469 CA: 128(10)113469q JOURNAL
Role of adhesion molecules in atherogenesis
AUTHOR(S): Yoshida, Masayuki
LOCATION: Med. Res. Inst., Tokyo Med. Dent. Univ., Tokyo, Japan, 101
JOURNAL: Domyaku Koka DATE: 1997 VOLUME: 25 NUMBER: 3 PAGES: 113-119
CODEN: DOMKDM ISSN: 0386-2682 LANGUAGE: Japanese PUBLISHER: Nippon
Domyaku Koka Gakkai

14/3/20 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

124339381 CA: 124(25)339381j JOURNAL
Adhesion molecules influencing atherosclerosis
AUTHOR(S): Tschoepe, D.
LOCATION: Diabetes Research Institute, Heinrich Heine University,
Duesseldorf, Germany, 40225
JOURNAL: Diabetes Res. Clin. Pract. DATE: 1996 VOLUME: 30 NUMBER:
Suppl. PAGES: S19-S24 CODEN: DRCPE9 ISSN: 0168-8227 LANGUAGE: English

14/3/21 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

118036695 CA: 118(5)36695z JOURNAL
Viral activation of the coagulation cascade
AUTHOR(S): Etingin, Orli R.; Silverstein, Roy L.; Hajjar, David P.
LOCATION: Med. Coll., Cornell Univ., New York, NY, 10021, USA
JOURNAL: Semin. Virol. DATE: 1992 VOLUME: 3 NUMBER: 2 PAGES: 125-33
CODEN: SEVIEL ISSN: 1044-5773 LANGUAGE: English
? ds

Set	Items	Description
S1	25	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHER-OSCLER?)
S2	14	RD S1 (unique items)
S3	13	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTENOSIS)
S4	9	RD S3 (unique items)
S5	2	S2 AND S4
S6	2	RD S5 (unique items)
S7	3360	RESTENOSIS AND ATHEROSCLEROSIS
S8	495	S7 AND REVIEW?
S9	303	RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10	0	S9 AND P(W)SELECTION?
S11	1	S9 AND P(W)SELECTIN?
S12	511	(RESTENOSIS OR ATHEROSCLEROSIS) AND P(W)SELECTIN?
S13	27	S12 AND REVIEW?
S14	21	RD S13 (unique items)

? s s12 and py=1994

511 S12
1920496 PY=1994
S15 18 S12 AND PY=1994
? rd s15

...completed examining records
S16 13 RD S15 (unique items)
? t s16/3/all

16/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09627100 BIOSIS NO.: 199598082018
Inhibition of platelet activity by S-nitrosoglutathione during coronary angioplasty.
AUTHOR: Langford E J; Brown A S; Wainwright R J; Debelder A J; Thomas M R; Smith R E A; Radomski M W; Martin J F(a); Moncada S
AUTHOR ADDRESS: (a)Cardiol. Dep., King's Coll. Hosp., London**UK
JOURNAL: Lancet (North American Edition) 344 (8935):p1458-1460 1994
ISSN: 0099-5355
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

16/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09566998 BIOSIS NO.: 199598021916
Role of **P-selectin** in animal models of thrombosis and **restenosis**.
AUTHOR: Shebuski Ronald J; Humphrey William R; Simmons Carol A; Hoover Jennifer L; Degraaf Garry L; Geng Jian G; Toombs Christopher F; Anderson Donald C
AUTHOR ADDRESS: Upjohn Lab., Kalamazoo, MI**USA
JOURNAL: Circulation 90 (4 PART 2):p1142 1994
CONFERENCE/MEETING: 67th Scientific Sessions of the American Heart Association Dallas, Texas, USA November 14-17, 1994
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

16/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09359869 BIOSIS NO.: 199497368239
Increase in the adhesion molecule **P-selectin** in endothelium
overlying atherosclerotic plaques: Coexpression with intercellular
adhesion molecule-1.
AUTHOR: Johnson-Tidey Ruth R; McGregor John L; Taylor Peter R; Poston Robin
N(a)
AUTHOR ADDRESS: (a)Dep. Experimental Pathology, UMDS, Med. Sch., 4th Floor,
Guy's Hosp., London Bridge, London SE1 **UK
JOURNAL: American Journal of Pathology 144 (5):p952-961 1994
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

16/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09226788 BIOSIS NO.: 199497235158
The protective role of high-density lipoprotein on
oxidized-low-density-lipoprotein-induced U937/endothelial cell
interactions.
AUTHOR: Maier Jeanette Anne Marie(a); Barenghi Livia; Pagani Franco;
Bradamante Silvia; Comi Paola; Ragnotti Giovanni
AUTHOR ADDRESS: (a)Dipartimento di Scienze Tecnol. Biomediche-Ospedale San
Raffaele, Via Olgettina 58, I-20132 Mila**Italy
JOURNAL: European Journal of Biochemistry 221 (1):p35-41 1994
ISSN: 0014-2956
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

16/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09156371 BIOSIS NO.: 199497164741
Purification of a novel cobra venom protease that cleaves the Von
Willebrand factor receptor on human platelets and the **P-**
selectin receptor on neutrophils.
AUTHOR: Andrews Robert K; Ward Christopher M; Dunlop Lindsay C; Berndt
Michael C
AUTHOR ADDRESS: Vascular Biol. Lab., Baker Med. Research Inst., Prahran
3181**Australia
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p294
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory
Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09156365 BIOSIS NO.: 199497164735
Expression cloning of a functional glycoprotein ligand for **P-**

selectin.

AUTHOR: Sako Dianne(a); Chang Xiao-Jia(a); Barone Karen M(a); Vachino Gloria(a); Shaw Gray(a); Veldman Trudi M(a); Bean Kevin M(a); Ahern Tim J (a); Furie Bruce; et al
AUTHOR ADDRESS: (a)Genetics Inst. Inc., 87 Cambridge Park Drive, Cambridge, MA 02140**USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p293
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09156358 BIOSIS NO.: 199497164728
Increase in **P-selectin** in the endothelium overlying human atherosclerotic plaques: Coexpression with ICAM-1.
AUTHOR: Johnson-Tidey Ruth R; Poston Robin N
AUTHOR ADDRESS: Dep. Experimental Pathol., UMDS, Guy's Hosp., London SE1 9RT**UK
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p291
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09156353 BIOSIS NO.: 199497164723
Structure/function studies of **P-selectin** glycoprotein ligand.
AUTHOR: Barone Karen M; Pittman Deborah; Shaw Gray
AUTHOR ADDRESS: Genetics Inst. Inc., 87 Cambridge Park Drive, Cambridge, MA 02140**USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p290
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09156280 BIOSIS NO.: 199497164650
Human endothelial cells treated by oxidized low density lipoproteins express **P-selectin** and bind monocytes.
AUTHOR: McGregor J L; Murphy J; Reck M-P; Gebuhrer V
AUTHOR ADDRESS: INSERM Unit 331, Fac. Med. Alexis Carrel, Pasteur Inst., Lyon**France
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p271
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994

ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/10 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

05875679 EMBASE No: 1994288635
Platelet alpha-granule release in cocaine users
Rinder H.M.; Ault K.A.; Jatlow P.I.; Kosten T.R.; Smith B.R.
Department of Laboratory Medicine, Yale University School of Medicine, PO
Box 208035, New Haven, CT 06520-8035 United States
Circulation (CIRCULATION) (United States) 1994, 90/3 (1162-1167)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

16/3/11 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

123310911 CA: 123(23)310911a CONFERENCE PROCEEDING
Endothelium, blood-born cells and intimal colony forming units in human
atherogenesis
AUTHOR(S): Balyasnikova, I. V.; Byzova, T. V.; Bystrevskaya, V. B.;
Ilyinskaya, O. P.; Krushinsky, A. V.; Popkova, V. M.; Romanov, Yu. A.;
Soboleva, E. L.; Tararak, E. M.; Smirnov, V. N.
LOCATION: Cardiology Research Center, Academy Medical Sciences, Moscow,
Russia,
JOURNAL: Eur. Sect. Meet., Int. Soc. Heart Res., 15th EDITOR: Haunsoe,
Stig (Ed), Kjeldsen, Keld (Ed), DATE: 1994 PAGES: 327-30 CODEN: 61SCA9
LANGUAGE: English PUBLISHER: Monduzzi Editore, Bologna, Italy

16/3/12 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

121026895 CA: 121(3)26895n PATENT
Cyclic peptide inhibitors of cellular adhesion derived from selectins
INVENTOR(AUTHOR): Heavner, George A.
LOCATION: USA
ASSIGNEE: Centocor, Inc.
PATENT: PCT International ; WO 9405310 A1 DATE: 940317
APPLICATION: WO 93US8504 (930908) *US 941653 (920908)
PAGES: 175 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-037/00A;
A61K-037/02B; C07K-005/00B; C07K-007/00B; C07K-015/00B; C07K-017/00B
DESIGNATED COUNTRIES: CA; JP; US DESIGNATED REGIONAL: AT; BE; CH; DE; DK
; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

16/3/13 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

120315824 CA: 120(25)315824x PATENT
P-selectin-derived inhibitors of leukocyte adhesion
INVENTOR(AUTHOR): Heavner, George A.; Epps, Leon A.
LOCATION: USA
ASSIGNEE: Centocor, Inc.
PATENT: PCT International ; WO 9405314 A1 DATE: 940317
APPLICATION: WO 93US7964 (930824) *US 941652 (920908)
PAGES: 56 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-037/02A;

C07K-007/00B DESIGNATED COUNTRIES: CA; JP DESIGNATED REGIONAL: AT; BE; CH
; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
? t sl6/kwic/all

>>>KWIC option is not available in file(s): 399

16/KWIC/1 (Item 1 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

1994

ABSTRACT: Platelet activation is associated with acute vessel occlusion and chronic **restenosis** after percutaneous transluminal coronary angioplasty (PTCA). Organic nitrates, which act by releasing the vasodilator and...

...of GSNO. Blood was sampled from the coronary sinus to measure platelet surface expression of **P-selectin** and glycoprotein IIb/IIIa as indices of platelet activation. In 7 control patients, PTCA caused a rise in platelet surface expression of **P-selectin** and glycoprotein IIb/IIIa, which was maximal 5 minutes after PTCA, indicating increased platelet activation...

...min before PTCA. GSNO significantly inhibited the PTCA-induced increase in platelet surface expression of **P-selectin** and glycoprotein IIb/IIIa without altering blood pressure. These findings show that platelets are activated...

16/KWIC/2 (Item 2 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Role of **P-selectin** in animal models of thrombosis and **restenosis**.

1994

16/KWIC/3 (Item 3 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Increase in the adhesion molecule **P-selectin** in endothelium overlying atherosclerotic plaques: Coexpression with intercellular adhesion molecule-1.

1994

ABSTRACT: **P-selectin** (GMP-140) is an adhesion molecule present within endothelial cells that is rapidly translocated to...

...mediates endothelial-leukocyte interactions. Immunohistochemical analysis of human atherosclerotic plaques has shown strong expression of **P-selectin** by the endothelium overlying active atherosclerotic plaques. **P-selectin** is not, however, detected in normal arterial endothelium or in endothelium overlying inactive fibrous plaques. Color image analysis was used to quantitate the degree of **P-selectin** expression in the endothelium and demonstrates a statistically significant increase in **P-selectin** expression by atherosclerotic endothelial cells. Double immunofluorescence shows that some of this **P-selectin** is expressed on the luminal surface of the endothelial cells. Previous work has demonstrated a...

...in atherosclerotic endothelium and a study on the expression of intercellular adhesion molecule-1 and **P-selectin** in **atherosclerosis** shows a highly positive correlation. These results suggest that the selective and cooperative expression of **P-selectin** and intercellular adhesion molecule-1 may be involved in the recruitment of monocytes into sites of **atherosclerosis**.

16/KWIC/4 (Item 4 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

1994

...ABSTRACT: novo protein synthesis. Interestingly, E-selectin, intercellular adhesion molecule-1, vascular cell-adhesion molecule or **P-selectin** induction was not apparent in this system suggesting the presence of an alternative system for...
MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**; ...

...**P-SELECTIN**;

16/KWIC/5 (Item 5 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

...cobra venom protease that cleaves the Von Willebrand factor receptor on human platelets and the **P-selectin** receptor on neutrophils.

1994

MISCELLANEOUS TERMS: **ATHEROSCLEROSIS**;

16/KWIC/6 (Item 6 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Expression cloning of a functional glycoprotein ligand for **P-selectin**.

1994

MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**;

16/KWIC/7 (Item 7 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Increase in **P-selectin** in the endothelium overlying human atherosclerotic plaques: Coexpression with ICAM-1.

1994

MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**;

16/KWIC/8 (Item 8 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Structure/function studies of **P-selectin** glycoprotein ligand.

1994

MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**;

16/KWIC/9 (Item 9 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Human endothelial cells treated by oxidized low density lipoproteins express **P-selectin** and bind monocytes.

1994

MISCELLANEOUS TERMS: **ATHEROSCLEROSIS**;

16/KWIC/10 (Item 1 from file: 73)
DIALOG(R)File 73:(c) 2001 Elsevier Science B.V. All rts. reserv.

...of circulating activated platelets in whole blood (those that express the alpha-granule membrane protein **P-selectin**), we found that 5 of 25 samples from 12 long-term cocaine users had a...

...at concentrations of 10sup -sup 7 to 10sup -sup 5 mol/L to cause platelet **P-selectin** expression in vitro in this study, coupled with the acute increase in circulating activated platelets...
 MEDICAL DESCRIPTORS:
 article; **atherosclerosis**--diagnosis--di; clinical article; controlled study; drug dependence--etiology--et; human; human cell; human tissue...
1994
 ? ds

Set	Items	Description
S1	25	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHER-OSCLER?)
S2	14	RD S1 (unique items)
S3	13	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTENOSIS)
S4	9	RD S3 (unique items)
S5	2	S2 AND S4
S6	2	RD S5 (unique items)
S7	3360	RESTENOSIS AND ATHEROSCLEROSIS
S8	495	S7 AND REVIEW?
S9	303	RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10	0	S9 AND P(W)SELECTION?
S11	1	S9 AND P(W)SELECTIN?
S12	511	(RESTENOSIS OR ATHEROSCLEROSIS) AND P(W)SELECTIN?
S13	27	S12 AND REVIEW?
S14	21	RD S13 (unique items)
S15	18	S12 AND PY=1994
S16	13	RD S15 (unique items)

? s s12 and restenosis

511 S12
 24623 RESTENOSIS
 S17 102 S12 AND RESTENOSIS
 ? s s17 and py=1994

102 S17
 1920496 PY=1994
 S18 4 S17 AND PY=1994
 ? rd s18

...completed examining records
 S19 2 RD S18 (unique items)
 ? t s19/3/all

19/3/1 (Item 1 from file: 5)
 DIALOG(R)File 5:Biosis Previews(R)
 (c) 2001 BIOSIS. All rts. reserv.

09627100 BIOSIS NO.: 199598082018
 Inhibition of platelet activity by S-nitrosoglutathione during coronary angioplasty.
 AUTHOR: Langford E J; Brown A S; Wainwright R J; Debelder A J; Thomas M R; Smith R E A; Radomski M W; Martin J F(a); Moncada S
 AUTHOR ADDRESS: (a)Cardiol. Dep., King's Coll. Hosp., London**UK
 JOURNAL: Lancet (North American Edition) 344 (8935):p1458-1460 **1994**
 ISSN: 0099-5355
 DOCUMENT TYPE: Article
 RECORD TYPE: Abstract
 LANGUAGE: English

19/3/2 (Item 2 from file: 5)
 DIALOG(R)File 5:Biosis Previews(R)

(c) 2001 BIOSIS. All rts. reserv.

09566998 BIOSIS NO.: 199598021916

Role of **P-selectin** in animal models of thrombosis and
restenosis.

AUTHOR: Shebuski Ronald J; Humphrey William R; Simmons Carol A; Hoover
Jennifer L; Degraaf Garry L; Geng Jian G; Toombs Christopher F; Anderson
Donald C

AUTHOR ADDRESS: Upjohn Lab., Kalamazoo, MI**USA

JOURNAL: Circulation 90 (4 PART 2):pI142 **1994**

CONFERENCE/MEETING: 67th Scientific Sessions of the American Heart
Association Dallas, Texas, USA November 14-17, 1994

ISSN: 0009-7322

RECORD TYPE: Citation

LANGUAGE: English

6/7/40 (Item 17 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

09288954 21026284 PMID: 11151785
Cardiac allograft vasculopathy--problem and model.
von Scheidt W
Medizinische Klinik und Poliklinik I Klinikum Grosshadern
Ludwig-Maximilians-Universitat Munchen 81366 Munchen, Germany.
wolfgang.scheidt@med1.med.uni-muenchen.de
Zeitschrift fur Kardiologie (Germany) 2000, 89 Suppl 9 pIX/2-5,
ISSN 0300-5860 Journal Code: 0360430
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Cardiac allograft vasculopathy (CAV) is an accelerated form of atherosclerosis induced by immunological endothelial injury with subsequent inflammatory repair responses in a milieu of additional nonimmunological risk factors. It is the leading cause of death beyond the first year after transplantation. The clinical situation is characterized by a poorly controlled complexity of pathogenetic and protective mechanisms and the heterogeneous nature concerning functional and structural manifestations, disease progression and prognosis. An early risk prediction algorithm for CAV is required in order to establish optimized preventive and therapeutical strategies. Experimental animals serve as model systems to selectively investigate different steps of the injury cascade providing specific insights into key mechanisms operating in CAV. Beyond its importance in transplantation medicine, human CAV can be taken as an unique model of atherosclerosis allowing evaluation and correlation of vascular function and morphology with the humoral and intracardiac activity/expression of mediators of the disease. Thus, CAV, beyond being a cumbersome clinical problem, represents an unique and attractive model of atherosclerosis in humans offering perspectives beyond the usual.
Record Date Created: 20010109
Record Date Completed: 20010412

6/7/41 (Item 18 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
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09263938 20584583 PMID: 11154223
Circulating autoantibodies to oxidized cardiolipin correlate with isoprostane F(2alpha)-VI levels and the extent of atherosclerosis in ApoE-deficient mice: modulation by vitamin E.
Pratico D; Tangirala R K; Horkko S; Witztum J L; Palinski W; FitzGerald G A
The Center for Experimental Therapeutics, Department of Pharmacology, University of Pennsylvania, Philadelphia, PA 19104, USA.
domenico@spirit.gcrc.upenn.edu
Blood (UNITED STATES) Jan 15 2001, 97 (2) p459-64, ISSN 0006-4971
Journal Code: 7603509
Contract/Grant No.: HL57505; HL; NHLBI; M01RR00040; RR; NCRR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Lipid peroxidation plays an important role in atherogenesis. Previous studies suggested that autoantibodies against epitopes of oxidized low-density lipoprotein may indicate the extent or rate of progression of

6/7/29 (Item 6 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
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11481077 98364976 PMID: 9701246

A mouse model of human familial hypercholesterolemia: markedly elevated low density lipoprotein cholesterol levels and severe atherosclerosis on a low-fat chow diet.

Powell-Braxton L; Veniant M; Latvala R D; Hirano K I; Won W B; Ross J; Dybdal N; Zlot C H; Young S G; Davidson N O

Cardiovascular Research, Genentech Inc., South San Francisco, California 94080, USA.

Nature medicine (UNITED STATES) Aug 1998, 4 (8) p934-8, ISSN 1078-8956 Journal Code: 9502015

Contract/Grant No.: DK 42086; DK; NIDDK; HL 18577; HL; NHLBI; HL 38180; HL; NHLBI; +

Comment in Nat Med. 1998 Aug;4(8) 899-900; Comment in PMID 9701240; Erratum in Nat Med 1998 Oct;4(10):1200

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Mutations in the low density lipoprotein (LDL) receptor gene cause familial hypercholesterolemia, a human disease characterized by premature atherosclerosis and markedly elevated plasma levels of LDL cholesterol and apolipoprotein (apo) B100. In contrast, mice deficient for the LDL receptor (Ldlr-/-) have only mildly elevated LDL cholesterol levels and little **atherosclerosis**. This difference results from extensive editing of the hepatic apoB mRNA in the mouse, which **limits** apoB100 synthesis in favor of apoB48 synthesis. We have generated Ldlr-/- mice that cannot edit the apoB mRNA and therefore synthesize exclusively apoB100. These mice had markedly elevated LDL cholesterol and apoB100 levels and developed extensive atherosclerosis on a chow diet. This authentic model of human familial hypercholesterolemia will provide a new tool for studying atherosclerosis.

Record Date Created: 19980825

Record Date Completed: 19980825

6/7/30 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11481071 98364970 PMID: 9701240
State of the art: **atherosclerosis** in a **limited** edition.
Rader D J; FitzGerald G A
Nature medicine (UNITED STATES) Aug 1998, 4 (8) p899-900, ISSN
1078-8956 Journal Code: 9502015
Comment on Nat Med. 1998 Aug;4(8) 934-8; Comment on PMID 9701246
Document type: Comment; News
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Record Date Created: 19980825
Record Date Completed: 19980825

Q4301. L554

14006752 BIOSIS NO.: 200300000781

Experimental **atherosclerosis**: A historical overview.

AUTHOR: Moghadasian Mohammed H(a)

AUTHOR ADDRESS: (a)Healthy Heart Program, St. Paul's Hospital, 180-1081

Burrard Street, Vancouver, BC, V6Z 1Y6, Canada**Canada E-Mail:

mhmoghad@interchange.ubc.ca

JOURNAL: Life Sciences 70 (8):p855-865 January 11 2002 2002

MEDIUM: print

ISSN: 0024-3205

DOCUMENT TYPE: Article; Literature Review

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Almost one-hundred years ago the first evidence of experimental **atherosclerosis** was reported. Over the past century, significant advances have been made in the development of animal models of human coronary artery disease. In this minireview, induction of atherosclerotic lesions in several animal models including rodents (mice, rabbits, rats, hamsters, guinea pigs), avian (pigeons, chickens, quail), swine, carnivora (dogs, cats), and non-human primates is discussed. The **limitations** and advantages of the animal models of **atherosclerosis** have been summarized. The transgenic/**knockout** animal models have greatly enhanced our understanding of **atherosclerosis**. Compared to wild-type counterparts, the knockout/transgenic animals develop atherogenesis faster without a need for a highly atherogenic diet. Although almost all investigations support a causal role for increased plasma cholesterol levels in the development of atherosclerotic vascular disease, an increasing body of evidence indicates serious involvement of other factors including oxidative stress, inflammation, infection and other emerging risk factors.

?

9/7/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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06921616 EMBASE No: 1997206086
Insights into selectin function from **knockout** mice
Frenette P.S.; Wagner D.D.
P.S. Frenette, Center for Blood Research, 800 Huntington Ave., Boston, MA
02114 United States
AUTHOR EMAIL: frenette@cbr.med.harvard.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 1997, 78/1
(60-64)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 42

RC633.T57

The development of animal models through gene targeting was very useful to the selectin field. Selectins are found on endothelium, platelets and leukocytes and, they mediate adhesion among these cell types. The removal of a single selectin gene taught us that **P-selectin** on the vessel wall mediates leukocyte rolling in the absence of inflammation and that all three selectins contribute to leukocyte rolling during inflammation. Similarly, **P-selectin** is responsible for early neutrophil recruitment while the other selectins contribute in later stages. The **knockout** animals also confirmed the important role of L-selectin in lymphocyte homing. Removal of both endothelial selectins uncovered the hidden importance of E-selectin in leukocyte homeostasis and showed that the endothelial selectins were as important for leukocyte extravasation as the leukocyte beta2 integrins. The submission of selectin-deficient mice to models of various human diseases can provide invaluable information on conditions in which an anti-selectin therapy may prove beneficial.

9/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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10163013 BIOSIS NO.: 199698617931

P-selectin knockout: A mouse model for various human diseases.

BOOK TITLE: Ciba Foundation Symposium; Cell adhesion and human disease

AUTHOR: Wagner Denisa D

BOOK AUTHOR/EDITOR: Marsh J; Goode J A: Eds

AUTHOR ADDRESS: Cent. Blood Res., Harvard Med. Sch., 800 Huntington Avenue, Boston, MA 02115**USA

JOURNAL: Ciba Foundation Symposium (198):p2-16 1995

BOOK PUBLISHER: John Wiley and Sons Ltd., Baffin Lane, Chichester PO 19 1UD, England

John Wiley and Sons, Inc., 605 Third Avenue, New York, New York 10158-0012, USA

CONFERENCE/MEETING: Symposium London, England, UK May 17-19, 1994

ISSN: 0300-5208 ISBN: 0-471-95279-6

RECORD TYPE: Citation

LANGUAGE: English

RB113 .037

6/7/23 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE
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07625908 EMBASE No: 1999112323
Experimental techniques and models in the study of the development and treatment of abdominal aortic aneurysm
Carrell T.W.G.; Smith A.; Burnand K.G.
T.W.G. Carrell, Academic Department of Surgery, St Thomas' Hospital, London SE1 7EH United Kingdom
British Journal of Surgery (BR. J. SURG.) (United Kingdom) 1999, 86/3 (305-312)
CODEN: BJSUA ISSN: 0007-1323
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 74

Background: It is still unclear what initiates aneurysmal dilatation and what determines whether or not an aneurysm will continue to expand and rupture. Early detection and operative repair of an abdominal aortic aneurysm (AAA) still remains the only effective means of reducing the high mortality rate associated with the condition. Endovascular techniques are being developed in an attempt to reduce the mortality rate associated with elective repair. A variety of animal models and experimental techniques have been described in the investigation of the pathophysiology of AAA and in the development of improved endovascular surgical and pharmacological therapies. This article discusses these models and techniques, their advantages and some of the problems encountered in extrapolating experimental findings to the human condition. Methods: This review is based on a search of the Medline database from 1966 to March 1998 using recognized key words and text words. A further search was then conducted on references quoted within selected relevant publications. Results and conclusion: Treatment of rodent aortas with intraluminal elastase or periaortic calcium chloride creates reproducible aneurysms that have certain similarities to the human pathology; such aneurysms have been favoured in the investigation of the pathophysiology of aneurysm expansion. However, these models lack several of the prominent features of the human lesion, such as **atherosclerosis** and intraluminal thrombosis. The development of gene **knockout** mice may lead to a more analogous aneurysm formation, with associated atherosclerosis. Many large animal models have been used in the development of endovascular techniques but, in general, these do not mimic the human pathophysiology and fail to predict medium- and long-term complications.

6/7/24 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14812134 22620038 PMID: 12588950
Hypercholesterolemia and changes in lipid and bile acid metabolism in male and female cyp7A1-deficient mice.
Erickson Sandra K; Lear Steven R; Deane Sean; Dubrac Sandrine; Huling Sandra L; Nguyen Lien; Bollineni Jaya S; Shefer Sarah; Hyogo Hideyuki; Cohen David E; Shneider Benjamin; Sehayek Ephraim; Ananthanarayanan Meena; Balasubramaniyan Natarajan; Suchy Fredrick J; Batta Ashok K; Salen Gerald
Department of Medicine, University of California, San Francisco, CA 94143.
Journal of lipid research (United States) 02 16 2003, 44 (5) p1001-9
, ISSN 0022-2275 Journal Code: 0376606
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM

Record type: In Process

Cholesterol 7 α -hydroxylase, a rate-limiting enzyme for bile acid synthesis, has been implicated in genetic susceptibility to **atherosclerosis**. The gene, CYP7A1, encoding a protein with this activity, is expressed normally only in hepatocytes and is highly regulated. Our cyp7A1 gene **knockout** mouse colony, as young adults on a chow diet, is hypercholesterolemic. These mice were characterized extensively to understand how cyp7A1 affects lipid and bile acid homeostasis in different tissue compartments and whether gender plays a modifying role. Both male and female cyp7A1-deficient mice had decreased hepatic LDL receptors, unchanged hepatic cholesterol synthesis, increased intestinal cholesterol synthesis and bile acid transporters, and decreased fecal bile acids but increased fecal sterols. In females, cyp7A1 deficiency also caused changes in hepatic fatty acid metabolism, decreased hepatic canalicular bile acid transporter, Bsep, and gallbladder bile composition altered to a lithogenic profile. Taken together, the data suggest that cyp7A1 deficiency results in a proatherogenic phenotype in both genders and leads to a prolithogenic phenotype in females.

Record Date Created: 20030507

6/7/25 (Item 2 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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14497882 22466635 PMID: 12578865

Reciprocal association of C-reactive protein with adiponectin in blood stream and adipose tissue.

Ouchi Noriyuki; Kihara Shinji; Funahashi Tohru; Nakamura Tadashi; Nishida Makoto; Kumada Masahiro; Okamoto Yoshihisa; Ohashi Koji; Nagaretani Hiroyuki; Kishida Ken; Nishizawa Hitoshi; Maeda Norikazu; Kobayashi Hideki; Hiraoka Hisatoyo; Matsuzawa Yuji

Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Suita, Osaka, Japan. ouchi@imed2.med.osaka-u.ac.jp

Circulation (United States) Feb 11 2003, 107 (5) p671-4, ISSN 1524-4539 Journal Code: 0147763

Document type: Clinical Trial; Controlled Clinical Trial; Journal Article
Languages: ENGLISH

Main Citation Owner: NLM

9/7/16 (Item 7 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
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09468946 21242601 PMID: 11344083

Direct viewing of **atherosclerosis** in vivo: plaque invasion by leukocytes is initiated by the endothelial selectins.

Eriksson E E; Xie X; Werr J; Thoren P; Lindbom L

Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden. einar.eriksson@fyfa.ki.se

FASEB journal - official publication of the Federation of American Societies for Experimental Biology (United States) May 2001, 15 (7) p1149-57, ISSN 0892-6638 Journal Code: 8804484

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Leukocyte infiltration in **atherosclerosis** has been extensively investigated by using histological techniques on fixed tissues. In this study, intravital microscopic observations of leukocyte recruitment in the aorta of atherosclerotic mice were performed. Interactions between leukocytes and atherosclerotic endothelium were highly transient, thereby limiting the ability for rolling leukocytes to firmly adhere. Leukocyte rolling was abolished by function inhibition of **P-selectin** ($P < 0.001$, $n = 8$), whereas antibody blockage of E-selectin ($n = 10$) decreased rolling leukocyte flux to $51 \pm 9.9\%$ (mean \pm SE, $P < 0.01$) and increased leukocyte rolling velocity to $162 \pm 18\%$ ($P < 0.01$) of pretreatment values. Notably, function inhibition of the integrin $\alpha(4)$ subunit ($n = 5$) had no effect on rolling flux ($107 \pm 25\%$, $P = 0.782$) or rolling velocity ($89 \pm 6.1\%$, $P = 0.147$), despite endothelial expression of vascular cell adhesion molecule 1 (VCAM-1). Leukocytes interacting with atherosclerotic endothelium were predominantly neutrophils, because treatment with antineutrophil serum decreased rolling and neutrophil counts in peripheral blood to the same extent. In conclusion, we present the first direct observations of **atherosclerosis** in vivo. We show that transient dynamics of leukocyte-endothelium interactions are important regulators of arterial leukocyte recruitment and that leukocyte rolling in **atherosclerosis** is critically dependent on the endothelial selectins. This experimental technique and the data presented introduce a novel perspective for the study of pathophysiological events involved in large-vessel disease.

/12 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

14599823 22402159 PMID: 12483207

Circulating activated platelets exacerbate **atherosclerosis** in mice deficient in apolipoprotein E.

Huo Yuqing; Schober Andreas; Forlow S Bradley; Smith David F; Hyman Matthew Craig; Jung Steffen; Littman Dan R; Weber Christian; Ley Klaus

Department of Biomedical Engineering and Cardiovascular Research Center, University of Virginia, Health Science Center, Charlottesville, Virginia, USA.

Nature medicine (United States) Jan 2003, 9 (1) p61-7, ISSN 1078-8956 Journal Code: 9502015

Contract/Grant No.: HL-58108; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

We studied whether circulating activated platelets and platelet-leukocyte aggregates cause the development of atherosclerotic lesions in apolipoprotein-E-deficient (ApoE(-/-)) mice. Circulating activated platelets bound to leukocytes, preferentially monocytes, to form platelet-monocyte/leukocyte aggregates. Activated platelets and platelet-leukocyte aggregates interacted with atherosclerotic lesions. The interactions of activated platelets with monocytes and atherosclerotic arteries led to delivery of the platelet-derived chemokines CCL5 (regulated on activation, normal T cell expressed and secreted, RANTES) and CXCL4 (platelet factor 4) to the monocyte surface and endothelium of atherosclerotic arteries. The presence of activated platelets promoted leukocyte binding of vascular cell adhesion molecule-1 (VCAM-1) and increased their adhesiveness to inflamed or atherosclerotic endothelium. Injection of activated wild-type, but not **P-selectin**-deficient, platelets increased monocyte arrest on the surface of atherosclerotic lesions and the size of atherosclerotic lesions in ApoE(-/-) mice. Our results indicate that circulating activated platelets and platelet-leukocyte/monocyte aggregates promote formation of atherosclerotic lesions. This role of activated platelets in **atherosclerosis** is attributed to platelet **P-selectin**-mediated delivery of platelet-derived proinflammatory factors to monocytes/leukocytes and the vessel wall.

Record Date Created: 20030106

9/7/11 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14864857 22617820 PMID: 12707243

Single injection of **P-selectin** or **P-selectin**
glycoprotein ligand-1 monoclonal antibody blocks neointima formation after
arterial injury in apolipoprotein E-deficient mice.

Phillips J William; Barringhaus Kurt G; Sanders John M; Hesselbacher Sean
E; Czarnik Ann C; Manka David; Vestweber Dietmar; Ley Klaus; Sarembock Ian
J

Department of Medicine, Cardiovascular Division, University of Virginia
Health System, Box 800158, Charlottesville, VA 22908-0158, USA.

Circulation (United States) 04 21 2003, 107 (17) p2244-9, ISSN
1524-4539 Journal Code: 0147763

Contract/Grant No.: HL-58108; HL; NHLBI; HL-66264; HL; NHLBI;
T32-HL-07355; HL; NHLBI

Comment in Circulation. 2003 May 6;107(17) 2175-7; Comment in PMID
12732592

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: Emerging data suggest that **P-selectin**, by
controlling adhesion of white blood cells, may be important in limiting the
response to vascular injury. METHODS AND RESULTS: We tested the hypothesis
that transient inhibition of **P-selectin** with either anti-
P-selectin monoclonal antibody (mAb) or anti-**P-**
selectin glycoprotein ligand-1 (PSGL-1) mAb would reduce neointima
formation in the setting of carotid denudation injury in
atherosclerosis-prone apolipoprotein E-/- mice. Neointima formation
at 28 days was reduced significantly, by 50% or 80%, by a single injection
on the day of injury of 100 or 200 microg **P-selectin** mAb RB
40.34 and by 55% by a single injection of 100 microg PSGL-1 mAb 4RA10 ($P <$
or $= 0.005$). In addition, there was a significant reduction in neointimal
macrophage content. CONCLUSIONS: These findings demonstrate that transient
P-selectin or PSGL-1 blockade at the time of arterial injury
significantly limits plaque macrophage content and neointima formation in a
dose-dependent manner after carotid denudation injury in apolipoprotein
E-/- mice.

Record Date Created: 20030506

Record Date Completed: 20030515

10 (Item 1 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

14883077 22528551 PMID: 12480714

Platelet **P-selectin** facilitates atherosclerotic lesion development.

Burger Peter C; Wagner Denisa D
Center for Blood Research and Department of Pathology, Harvard Medical School, Boston, MA 02115, USA.

Blood (United States) 12 12 2002, 101 (7) p2661-6, ISSN 0006-4971
Journal Code: 7603509

Contract/Grant No.: R01 HL 53756; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

P-selectin is an adhesion molecule expressed on activated platelets and endothelium. It is known to play an important role in **atherosclerosis**. **P-selectin** also circulates in plasma in a soluble form (sP-selectin), which induces procoagulant microparticle formation. We investigated the role of platelet versus endothelial **P-selectin** in generating sP-selectin and in the formation of atherosclerotic lesions in the apolipoprotein E (apoE)-deficient mouse model. For this we transplanted apoE(-/-)**P-selectin**(-/-) and apoE(-/-)**P-selectin**(+/+) lethally irradiated mice with bone marrow of either genotype. Seven months after transplantation, we determined from the chimeric animals that the majority of circulating sP-selectin was of endothelial origin. Thus, in **atherosclerosis**, the procoagulant sP-selectin reflects endothelial rather than platelet activation. We found that endothelial **P-selectin** was crucial for the promotion of atherosclerotic lesion growth because in its absence only relatively small lesions developed. However, platelet **P-selectin** also contributed to the lesion development because lesions in wild-type recipients receiving transplants with wild-type platelets were 30% larger than those receiving **P-selectin**-deficient platelets ($P < .008$) and were more frequently calcified (80% versus 44%). In comparison with **P-selectin** wild-type animals, absence of either endothelial or platelet **P-selectin** inhibited migration of smooth muscle cells into the lesion. Thus, in addition to endothelium, platelets and their **P-selectin** also actively promote advanced atherosclerotic lesion development.

Record Date Created: 20030318

knockout and (atherosclerosis) and p(w)selectin
53379 KNOCKOUT
158802 ATHEROSCLEROSIS
4181235 P
30362 SELECTIN
11513 P(W)SELECTIN
S8 19 KNOCKOUT AND (ATHEROSCLEROSIS) AND P(W)SELECTIN

? rd s18
>>>Set 18 has not yet been created.
? rd s8
...completed examining records
S9 16 RD S8 (unique items)
? t s9/7/all

9/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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14085730 BIOSIS NO.: 200300079759
Vascular and platelet expression of plasminogen activator inhibitor-1
contributes to the prothrombotic phenotype of apoE-knockout mice.
AUTHOR: Schaefer Katrin(a); Hecke Anneke(a); Mueller Katja(a); Goebel Julia
(a); Mounier Emmanuelle(a); Konstantinides Stavros(a)
AUTHOR ADDRESS: (a)Univ of Goettingen, Goettingen, Germany**Germany
JOURNAL: Circulation 106 (19 Supplement):pII-80 November 5 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions Chicago, IL, USA
November 17-20, 2002
SPONSOR: American Heart Association
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

9/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13889124 BIOSIS NO.: 200200517945
Deposition of platelet RANTES triggering monocyte recruitment requires
P-selectin and is involved in neointima formation after
arterial injury.
AUTHOR: Schober Andreas; Manka David; von Hundelshausen Philipp; Huo Yuqing
; Hanrath Peter; Sarembock Ian J; Ley Klaus; Weber Christian(a)
AUTHOR ADDRESS: (a)Kardiovaskulaere Molekularbiologie,
Universitaetsklinikum Aachen, Pauwelsstrasse 30, 52074, Aachen**Germany
E-Mail: cweber@ukaachen.de
JOURNAL: Circulation 106 (12):p1523-1529 September 17, 2002
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Chemokines expressed on atherosclerotic endothelium
or deposited by activated platelets have been implicated in monocyte
recruitment during atherogenesis and restenosis. Although the involvement
of P-selectin in these processes is evident from studies in
knockout mice, it has not been elucidated whether delivery of
platelet chemokines requires P-selectin, thus serving as a
P-selectin-dependent effector function. Methods and Results:
Using immunofluorescence and laminar flow assays, we found that the
deposition of the platelet-derived chemokine RANTES and monocyte arrest
subsequently triggered by RANTES immobilized on inflamed endothelium are

more efficient after preperfusion than after static preincubation of platelets and appear to depend on interactions of platelet but not endothelial **P-selectin**. This was revealed by the effects of **P-selectin** antibodies and comparison of **P-selectin**-deficient and wild-type platelets. Immunohistochemistry detected a substantial luminal expression of RANTES on neointimal lesions in wire-injured carotid arteries of apolipoprotein E (apoE)-deficient mice but not of mice with a combined deficiency in apoE and **P-selectin** (or platelet **P-selectin**). As assessed by histomorphometry, treatment of apoE-deficient mice with the RANTES receptor antagonist Met-RANTES markedly reduced neointimal plaque area and macrophage infiltration. Conclusions: Our data suggest that RANTES deposition and subsequent monocyte arrest are promoted by platelet **P-selectin** and involved in wire-induced intimal hyperplasia, and that blocking RANTES receptors attenuates neointima formation and macrophage infiltration. This mechanism represents an important component explaining the protection against neointimal growth in **P-selectin**-deficient mice and may represent a novel approach to the treatment of restenosis or **atherosclerosis** by the administration of chemokine receptor antagonists.

9/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13646022 BIOSIS NO.: 200200274843
A high-cholesterol diet leads to vascular inflammation and **atherosclerosis** in SR-BI deficient mice.
AUTHOR: Twisk Jaap(a); Van Eck Miranda(a); Bos Sophie(a); van Berkel Theo J C(a)
AUTHOR ADDRESS: (a)Leiden University/LACDR, Leiden**Netherlands
JOURNAL: Circulation 104 (17 Supplement):pII241-II242 October 23, 2001
MEDIUM: print
CONFERENCE/MEETING: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

9/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13634402 BIOSIS NO.: 200200263223
Peptide antagonists to **p-selectin**: Potential in anti-atherothrombotic therapy.
AUTHOR: Molenaar Tom J M(a); Twisk Jaap(a); Appeldoorn Chantal A M(a); de Haas Sonja A M(a); Michon Ingrid(a); van Berkel Theo J C(a); Kuiper Johan ; Biessen Erik A L
AUTHOR ADDRESS: (a)Leiden Univ, Leiden**Netherlands
JOURNAL: Circulation 104 (17 Supplement):pII38-II39 October 23, 2001
MEDIUM: print
CONFERENCE/MEETING: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

9/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

13329489 BIOSIS NO.: 200100536638
Adhesion molecules and atherogenesis.
AUTHOR: Huo Y; Ley K(a)
AUTHOR ADDRESS: (a)Department of Biomedical Engineering, Health Science
Center, University of Virginia, Charlottesville, VA, 22908**USA
JOURNAL: Acta Physiologica Scandinavica 173 (1):p35-43 September, 2001
MEDIUM: print
ISSN: 0001-6772
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: **Atherosclerosis** is an inflammatory disease of the vessel wall characterized by monocyte infiltration in response to pro-atherogenic factors such as oxidized lipids. Recently, the role of specific adhesion molecules in this process has been explored. The endothelium overlying atherosclerotic lesions expresses **P-selectin** and the shoulder regions express vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1), which is also expressed on endothelium in regions not prone to plaque development. Serum levels of soluble **P-selectin**, ICAM-1 and VCAM-1 are elevated in patients with angina pectoris or peripheral atherosclerotic disease. Reconstituted in vitro systems using monocytes on cytokine-activated endothelial cells under shear flow suggested the involvement of **P-selectin**, L-selectin, VCAM-1, its ligand, VLA-4 integrin and CD18 integrins. Studies of monocyte adhesion in isolated perfused carotid arteries harvested from atherosclerotic (apoE^{-/-}) mice show a predominant involvement of **P-selectin** and its ligand **P-selectin** glycoprotein-1 (PSGL-1) in rolling and of VLA-4 and VCAM-1 in firm adhesion. Consistent with these findings, apoE^{-/-} mice that are also deficient for **P-selectin** show significantly reduced atherosclerotic lesion sizes and are almost completely protected from neointimal growth after vascular injury. Milder effects are also seen in the low-density lipoprotein (LDL) receptor deficient (LDLR^{-/-}) mouse. In a high cholesterol/cholate model, a role of ICAM-1 and CD18 integrins was also shown, but this awaits confirmation in more physiologic models. Transient blockade of the VLA-4/VCAM-1 adhesion pathway by antibodies or peptides in apoE^{-/-} or LDLR^{-/-} mice reduced monocyte and lipid accumulation in lesions. These data suggest that **P-selectin**, PSGL-1, VLA-4 and VCAM-1 are the most important adhesion molecules involved in monocyte recruitment to atherosclerotic lesions.

9/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12904893 BIOSIS NO.: 200100112042
Expression of SR-B1 in atherosclerotic lesions from apoE-deficient mice is inversely correlated with the severity of lesion development.
AUTHOR: Twisk Jaap(a); Van Berkel Theo J C(a)
AUTHOR ADDRESS: (a)Leiden/Amsterdam Ctr for Drug Research, Leiden**
Netherlands
JOURNAL: Circulation 102 (18 Supplement):pII48 October 31, 2000
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

9/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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11909303 BIOSIS NO.: 199900155412
Donor and recipient contributions of ICAM-1 and **P-selectin** in
parenchymal rejection and graft arteriosclerosis: Insights from double
knockout mice.
AUTHOR: Raisanen-Sokolowski A K; Glysing-Jensen T; Russell M E
AUTHOR ADDRESS: Cardiovascular Biol. Lab., Harvard Sch. Public Health,
Brigham and Women's Hosp., Harvard Med. Sch.,**USA
JOURNAL: Journal of Heart and Lung Transplantation 18 (1):p61 Jan., 1999
CONFERENCE/MEETING: Nineteenth Annual Meeting and Scientific Sessions of
the International Society for Heart and Lung Transplantation San
Francisco, California, USA April 21-24, 1999
SPONSOR: International Society for Heart and Lung Transplantation
ISSN: 1053-2498
RECORD TYPE: Citation
LANGUAGE: English

9/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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10163013 BIOSIS NO.: 199698617931
P-selectin knockout: A mouse model for various human
diseases.
BOOK TITLE: Ciba Foundation Symposium; Cell adhesion and human disease
AUTHOR: Wagner Denisa D
BOOK AUTHOR/EDITOR: Marsh J; Goode J A: Eds
AUTHOR ADDRESS: Cent. Blood Res., Harvard Med. Sch., 800 Huntington
Avenue, Boston, MA 02115**USA
JOURNAL: Ciba Foundation Symposium (198):p2-16 1995
BOOK PUBLISHER: John Wiley and Sons Ltd., Baffin Lane, Chichester PO 19
1UD, England
John Wiley and Sons, Inc., 605 Third Avenue, New York, New
York 10158-0012, USA
CONFERENCE/MEETING: Symposium London, England, UK May 17-19, 1994
ISSN: 0300-5208 ISBN: 0-471-95279-6
RECORD TYPE: Citation
LANGUAGE: English

9/7/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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06921616 EMBASE No: 1997206086
Insights into selectin function from **knockout** mice
Frenette P.S.; Wagner D.D.
P.S. Frenette, Center for Blood Research, 800 Huntington Ave., Boston, MA
02114 United States
AUTHOR EMAIL: frenette@cbr.med.harvard.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 1997, 78/1
(60-64)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 42

The development of animal models through gene targeting was very useful

to the selectin field. Selectins are found on endothelium, platelets and leukocytes and, they mediate adhesion among these cell types. The removal of a single selectin gene taught us that **P-selectin** on the vessel wall mediates leukocyte rolling in the absence of inflammation and that all three selectins contribute to leukocyte rolling during inflammation. Similarly, **P-selectin** is responsible for early neutrophil recruitment while the other selectins contribute in later stages. The **knockout** animals also confirmed the important role of L-selectin in lymphocyte homing. Removal of both endothelial selectins uncovered the hidden importance of E-selectin in leukocyte homeostasis and showed that the endothelial selectins were as important for leukocyte extravasation as the leukocyte beta2 integrins. The submission of selectin-deficient mice to models of various human diseases can provide invaluable information on conditions in which an anti-selectin therapy may prove beneficial.

9/7/10 (Item 1 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
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14883077 22528551 PMID: 12480714

Platelet **P-selectin** facilitates atherosclerotic lesion development.

Burger Peter C; Wagner Denisa D
Center for Blood Research and Department of Pathology, Harvard Medical School, Boston, MA 02115, USA.

Blood (United States) 12 12 2002, 101 (7) p2661-6, ISSN 0006-4971
Journal Code: 7603509

Contract/Grant No.: R01 HL 53756; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

P-selectin is an adhesion molecule expressed on activated platelets and endothelium. It is known to play an important role in **atherosclerosis**. **P-selectin** also circulates in plasma in a soluble form (sP-selectin), which induces procoagulant microparticle formation. We investigated the role of platelet versus endothelial **P-selectin** in generating sP-selectin and in the formation of atherosclerotic lesions in the apolipoprotein E (apoE)-deficient mouse model. For this we transplanted apoE(-/-)**P-selectin**(-/-) and apoE(-/-)**P-selectin**(+/+) lethally irradiated mice with bone marrow of either genotype. Seven months after transplantation, we determined from the chimeric animals that the majority of circulating sP-selectin was of endothelial origin. Thus, in **atherosclerosis**, the procoagulant sP-selectin reflects endothelial rather than platelet activation. We found that endothelial **P-selectin** was crucial for the promotion of atherosclerotic lesion growth because in its absence only relatively small lesions developed. However, platelet **P-selectin** also contributed to the lesion development because lesions in wild-type recipients receiving transplants with wild-type platelets were 30% larger than those receiving **P-selectin**-deficient platelets (P < .008) and were more frequently calcified (80% versus 44%). In comparison with **P-selectin** wild-type animals, absence of either endothelial or platelet **P-selectin** inhibited migration of smooth muscle cells into the lesion. Thus, in addition to endothelium, platelets and their **P-selectin** also actively promote advanced atherosclerotic lesion development.

Record Date Created: 20030318

Record Date Completed: 20030521

9/7/11 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
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14864857 22617820 PMID: 12707243

Single injection of **P-selectin** or **P-selectin** glycoprotein ligand-1 monoclonal antibody blocks neointima formation after arterial injury in apolipoprotein E-deficient mice.

Phillips J William; Barringhaus Kurt G; Sanders John M; Hesselbacher Sean E; Czarnik Ann C; Manka David; Vestweber Dietmar; Ley Klaus; Sarembock Ian J

Department of Medicine, Cardiovascular Division, University of Virginia Health System, Box 800158, Charlottesville, VA 22908-0158, USA.

Circulation (United States) 04 21 2003, 107 (17) p2244-9, ISSN 1524-4539 Journal Code: 0147763

Contract/Grant No.: HL-58108; HL; NHLBI; HL-66264; HL; NHLBI; T32-HL-07355; HL; NHLBI

Comment in Circulation. 2003 May 6;107(17) 2175-7; Comment in PMID 12732592

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: Emerging data suggest that **P-selectin**, by controlling adhesion of white blood cells, may be important in limiting the response to vascular injury. **METHODS AND RESULTS:** We tested the hypothesis that transient inhibition of **P-selectin** with either anti-**P-selectin** monoclonal antibody (mAb) or anti-**P-selectin** glycoprotein ligand-1 (PSGL-1) mAb would reduce neointima formation in the setting of carotid denudation injury in **atherosclerosis**-prone apolipoprotein E-/- mice. Neointima formation at 28 days was reduced significantly, by 50% or 80%, by a single injection on the day of injury of 100 or 200 microg **P-selectin** mAb RB 40.34 and by 55% by a single injection of 100 microg PSGL-1 mAb 4RA10 ($P < 0.005$). In addition, there was a significant reduction in neointimal macrophage content. **CONCLUSIONS:** These findings demonstrate that transient **P-selectin** or PSGL-1 blockade at the time of arterial injury significantly limits plaque macrophage content and neointima formation in a dose-dependent manner after carotid denudation injury in apolipoprotein E-/- mice.

Record Date Created: 20030506

Record Date Completed: 20030515

9/7/12 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
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14599823 22402159 PMID: 12483207

Circulating activated platelets exacerbate **atherosclerosis** in mice deficient in apolipoprotein E.

Huo Yuqing; Schober Andreas; Forlow S Bradley; Smith David F; Hyman Matthew Craig; Jung Steffen; Littman Dan R; Weber Christian; Ley Klaus

Department of Biomedical Engineering and Cardiovascular Research Center, University of Virginia, Health Science Center, Charlottesville, Virginia, USA.

Nature medicine (United States) Jan 2003, 9 (1) p61-7, ISSN 1078-8956 Journal Code: 9502015

Contract/Grant No.: HL-58108; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

We studied whether circulating activated platelets and platelet-leukocyte aggregates cause the development of atherosclerotic lesions in

apolipoprotein-E-deficient (ApoE(-/-)) mice. Circulating activated platelets bound to leukocytes, preferentially monocytes, to form platelet-monocyte/leukocyte aggregates. Activated platelets and platelet-leukocyte aggregates interacted with atherosclerotic lesions. The interactions of activated platelets with monocytes and atherosclerotic arteries led to delivery of the platelet-derived chemokines CCL5 (regulated on activation, normal T cell expressed and secreted, RANTES) and CXCL4 (platelet factor 4) to the monocyte surface and endothelium of atherosclerotic arteries. The presence of activated platelets promoted leukocyte binding of vascular cell adhesion molecule-1 (VCAM-1) and increased their adhesiveness to inflamed or atherosclerotic endothelium. Injection of activated wild-type, but not P-selectin-deficient, platelets increased monocyte arrest on the surface of atherosclerotic lesions and the size of atherosclerotic lesions in ApoE(-/-) mice. Our results indicate that circulating activated platelets and platelet-leukocyte/monocyte aggregates promote formation of atherosclerotic lesions. This role of activated platelets in atherosclerosis is attributed to platelet P-selectin-mediated delivery of platelet-derived proinflammatory factors to monocytes/leukocytes and the vessel wall.

Record Date Created: 20030106

Record Date Completed: 20030318

9/7/13 (Item 4 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
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10095398 22052265 PMID: 12057918

Atherosclerotic lesions grow through recruitment and proliferation of circulating monocytes in a murine model.

Lessner Susan M; Prado Heather L; Waller Edmund K; Galis Zorina S
Division of Cardiology, Emory University School of Medicine, Atlanta, Georgia 30322, USA.

American journal of pathology (United States) Jun 2002, 160 (6)
p2145-55, ISSN 0002-9440 Journal Code: 0370502

Contract/Grant No.: 5 T32 HL07745 07; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

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Macrophage-derived foam cells in developing atherosclerotic lesions may potentially originate either from recruitment of circulating monocytes or from migration of resident tissue macrophages. In this study, we have determined the source of intimal macrophages in the apoE-knockout mouse flow-cessation/hypercholesterolemia model of atherosclerosis using a bone marrow transplantation approach. We also examined the time course and spatial distribution of intercellular adhesion molecule-1 and vascular cell adhesion molecule-1 expression to assess whether endothelial adhesion molecules were involved in recruitment of either circulating monocytes or resident macrophages. We used allelic variants of the mouse common leukocyte antigen (CD45) to distinguish host-derived and donor-derived white blood cells (WBCs) both in blood and in macrophage-rich carotid lesions. We found that the distribution of CD45 isoforms in lesions is similar to that of circulating WBCs, whereas the host-type CD45 isoform is more prevalent in resident adventitial macrophages. These data indicate that macrophage-derived foam cells in the lesion derive mainly from circulating precursors rather than from resident macrophages. The corresponding time course of intercellular adhesion molecule-1 and vascular cell adhesion molecule-1 expression suggests that recruitment of circulating WBCs by endothelial adhesion molecules is likely to be more important during lesion initiation than during the later phase of rapid lesion growth.

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09689155 21481241 PMID: 11597929

S17834, a new inhibitor of cell adhesion and **atherosclerosis** that targets nadph oxidase.

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Arteriosclerosis, thrombosis, and vascular biology (United States) Oct 2001, 21 (10) p1577-84, ISSN 1524-4636 Journal Code: 9505803

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microdant stress is involved in the events that accompany endothelial cell expression of adhesion molecules and leukocyte adherence in many disease states, including **atherosclerosis**. A recently discovered benzo(b)pyran-4-one derivative, S17834 (10 to 50 micromol/L), reduced tumor necrosis factor-stimulated vascular cell adhesion molecule-1 (VCAM) mRNA accumulation and protein expression in human umbilical vein endothelial cells. Intercellular cell adhesion molecule-1 and E-selectin were also inhibited by S17834, but platelet endothelial cell adhesion molecule-1 was not. Adherence of U937 monocytic cells to the endothelial cells as well as to plastic plates coated with soluble VCAM, intercellular cell adhesion molecule-1, **P-selectin**, and E-selectin was also decreased.

Consistent with an antioxidant mechanism of action, S17834 (10 to 50 micromol/L) inhibited tumor necrosis factor-stimulated release of superoxide from endothelial cells measured by cytochrome c reduction. S17834 had no effect on superoxide produced by xanthine oxidase, indicating that rather than by acting as a scavenger of superoxide anion, the drug acts by inhibiting the production of free radicals. Indeed, S17834 inhibited NADPH oxidase activity of endothelial cell membranes. The ability to inhibit superoxide anion production appears to be key in the effect of S17834 on superoxide anion production and VCAM expression, because these actions were mimicked by adenovirus-mediated overexpression of superoxide dismutase. Furthermore, these actions may be relevant in vivo, because S17834 reduced aortic superoxide anion levels by 40% and aortic atherosclerotic lesions by 60% in apolipoprotein E-deficient mice. These results indicate that S17834 inhibits adhesion molecule expression and adherence of leukocytes to endothelial cells as well as aortic atherogenesis and that perhaps these effects can be explained by its ability to inhibit endogenous superoxide anion production.

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Localized reduction of **atherosclerosis** in von Willebrand factor-deficient mice.

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Blood (United States) Sep 1 2001, 98 (5) p1424-8, ISSN 0006-4971
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To examine the role of the platelet adhesion molecule von Willebrand factor (vWf) in atherogenesis, vWf-deficient mice (vWf^{-/-}) were bred with mice lacking the low-density lipoprotein receptor (LDLR^{-/-}) on a C57BL/6J background. LDLR^{-/-}-vWf^{+/+} and LDLR^{-/-}-vWf^{-/-} mice were placed on a diet rich in saturated fat and cholesterol for different lengths of time. The atherogenic diet stimulated leukocyte rolling in the mesenteric venules in both genotypes, indicating an increase in P-selectin-mediated adhesion to the endothelium. After 8 weeks on the atherogenic diet, the fatty streaks formed in the aortic sinus of LDLR^{-/-}-vWf^{-/-} mice of either sex were 40% smaller and contained fewer monocytes than those in LDLR^{-/-}-vWf^{+/+} mice. After 22 weeks on the atherogenic diet (early fibrous plaque stage), the difference in lesion size in the aortic sinus persisted. Interestingly, the lesion distribution in the aortas of LDLR^{-/-}-vWf^{-/-} animals was different from that of LDLR^{-/-}-vWf^{+/+} animals. In vWf-positive mice, half of all lesions were located at the branch points of the renal and mesenteric arteries, whereas lesions in this area were not as prominent in the vWf-negative mice. These results indicate that the absence of vWf primarily affects the regions of the aorta with disturbed flow that are prone to atherosclerosis. Thus, vWf may recruit platelets/leukocytes to the lesion in a flow-dependent manner or may be part of the mechano-transduction pathway regulating endothelial response to shear stress.

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Direct viewing of atherosclerosis in vivo: plaque invasion by leukocytes is initiated by the endothelial selectins.

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Leukocyte infiltration in atherosclerosis has been extensively investigated by using histological techniques on fixed tissues. In this study, intravital microscopic observations of leukocyte recruitment in the aorta of atherosclerotic mice were performed. Interactions between leukocytes and atherosclerotic endothelium were highly transient, thereby limiting the ability for rolling leukocytes to firmly adhere. Leukocyte rolling was abolished by function inhibition of P-selectin (P<0.001, n=8), whereas antibody blockage of E-selectin (n=10) decreased rolling leukocyte flux to 51 +/- 9.9% (mean +/- SE, P<0.01) and increased leukocyte rolling velocity to 162 +/- 18% (P<0.01) of pretreatment values. Notably, function inhibition of the integrin alpha(4) subunit (n=5) had no effect on rolling flux (107 +/- 25%, P=0.782) or rolling velocity (89 +/- 6.1%, P=0.147), despite endothelial expression of vascular cell adhesion molecule 1 (VCAM-1). Leukocytes interacting with atherosclerotic endothelium were

predominantly neutrophils, because treatment with antineutrophil serum decreased rolling and neutrophil counts in peripheral blood to the same extent. In conclusion, we present the first direct observations of **atherosclerosis** in vivo. We show that transient dynamics of leukocyte-endothelium interactions are important regulators of arterial leukocyte recruitment and that leukocyte rolling in **atherosclerosis** is critically dependent on the endothelial selectins. This experimental technique and the data presented introduce a novel perspective for the study of pathophysiological events involved in large-vessel disease.

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